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EXPERIMENTS ON TEMPORARY OBSTRUCTION OF THE INTERNAL AUDITORY ARTERY.*†‡

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Certain types of sudden deafness are commonly considered to be the result of a vascular phenomenon in the inner ear and not infrequently associated with a considerable degree of recovery.

There is little factual information regarding these clinical observations; however, animal experiments indicate that the cochlea is exceedingly sensitive to its oxygen supply and when the oxygen supply is cut off electrical activity deteriorates within seconds.[1] Experiments have shown that the vascular system of the inner ear is vital for the continuous supply of oxygen and metabolites. Permanent obstruction of the inferior cochlear vein and its tributaries, and permanent occlusion of the internal auditory artery, produce rapid loss of function and characteristic histologic damage.[2,3,4] How the

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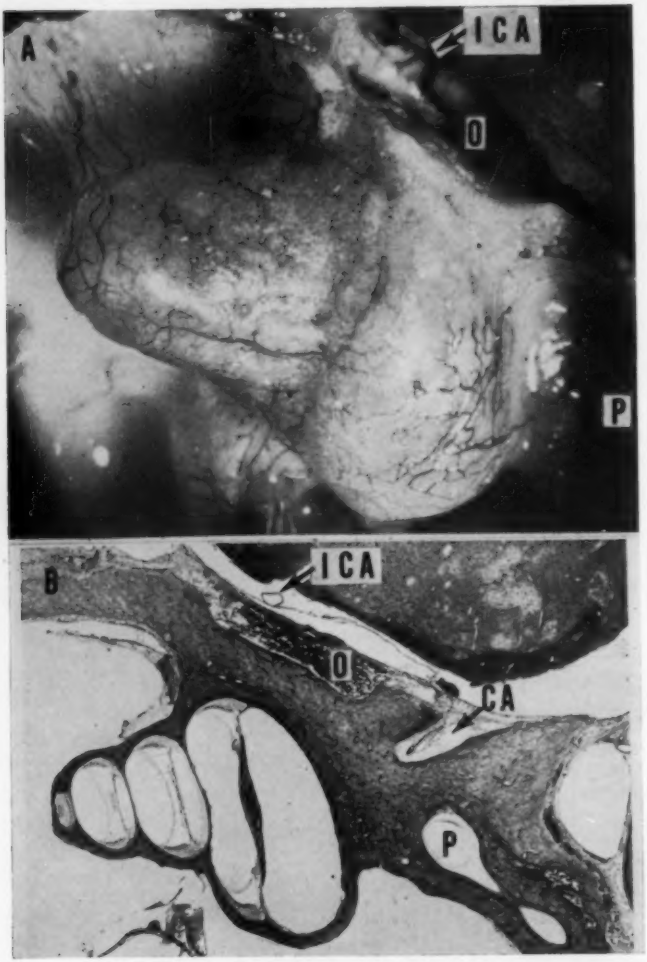

Fig. 1. a.—Enlarged photograph of the surgical field (O) showing the location of the inferior cerebellar artery (ICA) in relation to the cochlea. b.—Photomicrograph through a comparable area showing these relations on cross section. The surgical defect (O) is filled with gelfoam. CA is the cochlear aqueduct. P is the posterior ampulla.

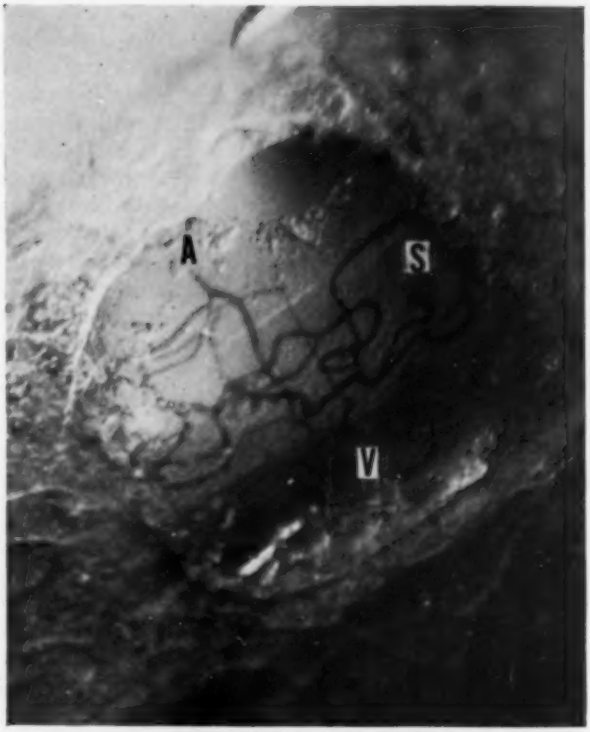

Fig. 2. Photograph through the microscope showing blood vessels exposed by removing cochlear capsule. A.—Radiating arterioie; S.—Stria capillaries; V.—Collecting venules.

cochlea withstands temporary interruptions in its blood supply is not known. The surgical accessibility of the arterial branch to the inner ear in the guinea pig permitted an investigation on the effect of temporary obstruction of the artery (see Fig. 1). This was monitored continuously by observation of the cochlear blood flow in the apical turn, either by fenestrating or merely thinning the capsule. Obstruction was produced for various lengths of time by pressure with a probe mounted in a micromanipulator. Return of flow after release of pressure terminated a successful experiment. The details of the

surgical approach have been described in a previous report.[3] A complete or almost complete cessation of blood flow in the cochlea was produced for one to 120 minutes and the results studied in three ways. The changes in the exposed vascular bed in the fenestrated cochlea were studied with the aid of moving pictures taken at magnifications up to 275X (see Fig. 2); the changes in function were assessed by cochlear microphonics and action potential recordings; and finally the inner ears were examined histologically. Different animals with comparable times of obstruction were used for each series of observations. In the groups studied histologically, the animals were kept alive for various periods of time up to three months. In contrast to permanent obstruction, vestibular symptoms were minimal or absent in these animals recovering from temporary arterial obstruction.

FINDINGS.

A. Changes in blood flow of the stria vascularis and spiral ligament: The vascular pattern and the nature of the normal blood flow in the fenestrated apical area of the guinea pig cochlea has been described previously. Regularity of flow and absence of diameter changes are striking. When the probe is successfully applied on the ventral surface of the brain stem over the branch of the inferior cerebellar artery cochlear, blood flow stops almost immediately. Sometimes a little pulsatile forward movement is observed with each inspiration, or a very slow continuous flow is seen. With the micromanipulator, further pressure usually succeeds in stopping the movement. The slow flow in the strial vessels is the most easily stopped. The blood cells usually remain in all the exposed vessels; however, with long obstruction some branches may become partly or completely free of cells, yet remain open and apparently filled with plasma. "Critical closure" of arterioles with falling pressure, described by Burton, was not seen in our experiment even when pressure and flow were absent. There is no evidence of clotting or dissolution of cells or movement of cells through the vessel wall. There is little or no change in diameter of the vessels in the fenestra. The blood becomes darker. There is no sticking of blood cells to the vessel wall during short periods of obstruction. With release

of pressure , flow returns at once, and immediately after return of flow white blood cells sometimes adhere to the wall of the strial vessels but not to the wall of arteriole or arteriovenous arcade where flow is fast. Larger numbers of white masses of various sizes flow through the vessels at this time. Flow is much more rapid than normal for about 30 to 180 seconds. As flow rate returns to normal, the vessels resume their normal appearance, but sometimes a branch of the stria may not resume flow. With longer periods of obstruction, 30 to 60 mintes, changes in the vascular contents take place while the blood is stagnant, which are made evident with resumption of blood flow. These are localized accumulations of blood cells and platelets adherent to the vessel wall and to each other. With return of supernormal flow these elements are rapidly washed away as normal flow and a normal lumen is restored. The restoration of normal blood flow, even after 60 minutes of obstruction, was the usual finding. Occasionally flow did not return on release of the probe. This was considered due to irreversible damage (thrombosis) of the vessel under the probe. In this area, some changes in diameter due to local trauma were consistently observed. Localized narrowing of the vessel was sometimes seen during manipulation, and dilation followed release of the pressure. This dilation was considered to be responsible, at least in part, for the short but consistent supernormal flow seen at the fenestration site; however, there are many factors besides local trauma to be considered when blood flow is stopped in this manner. The accumulation of platelets adhering to the vessel wall and to one another at the site of trauma has been observed by Zweifach,[5] and by Lutz, Fulton and Akers.[6] Finally, the vessels were seen to become completely plugged with red and white cells. Lutz, Fulton and Akers observe platelets throughout the field adhering to the vessel wall after 20 to 45 minutes of obstruction. On release of obstruction circulating erythrocytes made channels through it, and platelet emboli were set free. This corresponds to our observations in the cochlea. Some of the white masses that flow into the field may arise from the site of pressure. They also report that erythrocytes may remain unagglutinated even after many hours of stasis.

Denny-Brown and Meyer[7] produced temporary (three and

one-half minutes) cortical ischemia by occlusion of the middle cerebral artery and observed failure of the electrical activity within a few seconds, which recovered rapidly and completely on release of obstruction without damage to the vessels. They found that venous stasis after prolonged (15 to 30 minutes) ischemia could be reversed by elevating the blood pressure. When blood flow in the middle cerebral artery was stopped for longer periods irreversible damage to vascular endothelium led to infarction. They report that dilation, segmentation and stasis in the terminal venules was the first event following occlusion. This was followed by endothelial damage of the venules. They observed small microemboli lodging in the cortical vessels after prolonged occlusion and release of the middle cerebral artery. These were grey white and lodged temporarily in arteries of 50 to 150 micra in diameter. There was no arterial spasm at the site of lodgment. The microemboli were seen to move gradually to the terminal ramifications and in two to four minutes fragmented into minute particles and disappeared.

Zweifach notes vasodilation beyond the site of local trauma. We did not observe dilation in the region fenestrated during manipulation of the probe, but the slight dilation of the vessels in this terminal vascular bed, sometimes seen on return of blood flow, may have been due in part to the effect of anoxia and accumulation of CO_2 and acid metabolites on the vessel wall independent of the vessel changes at the point of obstruction. Bean and his associates[8,9] have described this effect in intestinal vessels. In the cortex, Meyer, Fang and Denny-Brown[10] found that on release from obstruction, local cortical O_2 tension rises along with temperature and persists for one to five minutes depending upon the severity of ischemia, before returning to a steady state. This supernormal phase was considered due to local reactive hyperemia. Since increased local acidity of tissue (P_h) was recorded throughout this response, acidity rather than anoxia was considered the effective local dilator. Our observations on the cochlea during general asphyxia indicate that there is a gradual reduction in flow without change in vessel lumen or white blood cell sticking; however, occasional loss of red blood cells into the

surrounding tissue is noted on recovery from asphyxia and here, too, flow returns to normal after going through a supernormal phase. Control studies in our experiments indicated that blood pressure in the carotid was not affected by these manipulations on the artery, so that this was not a factor to be considered in explaining the supernormal flow immediately on release of obstruction. In general, however, blood pressure has perhaps the most important single influence on the cerebral circulation. Unlike the findings of Meyer[11,12] on obstruction of the middle cerebral artery, perivascular edema or diapedesis of blood cells was not seen in the fenestrated area. No evidence of rupture of a blood vessel due to weakening of the vessel wall was found. Meyer quotes Bayliss that pressure within the vessel may act as a stimulus to its smooth muscle cells. With reduced intraluminal pressure these may relax to dilate and with increased pressure may contract to narrow the vessel. We did not observe these changes, but realize that the cochlear vessels exposed by the fenestra have little or no smooth muscle.

To summarize this portion of the experiment, one may say that cochlear blood flow may return to normal even after one hour of stasis, and that even the dislodged platelet masses appear to be washed through without plugging the vessels. The blood flow rate is greater than normal immediately after release from obstruction. The internal auditory artery appears to be the sole supply to the cochlea since pressure upon it results in complete cessation of blood flow.

B. Changes in function of the cochlea as revealed in evoked microphonics and action potentials: The sudden and complete occlusion of the arterial supply to the inner ear produces profound and rapid changes in cochlear responses that give insight into the oxygen requirements of the cells generating them. These findings, expressed as a function of time, may be compared with those in other tissues of the nervous system as reported by Gerard.[13]

Method. These experiments were carried out in 16 guinea pigs anesthetized with Dial® in urethane (0.5 cc. per kilo body weight). The operative approach and insertion of silver

wire electrodes in the first turn of the cochlea have been described elsewhere.[14,15] The surgical approach for the vascular supply of the inner ear and technique of occlusion have been described previously. The sound stimuli used in these experiments were 8,000 or 10,000 cycle tone pips. The intensity was at high level, near the point of maximum response of cochlear microphonics. The duration of the tone-pip

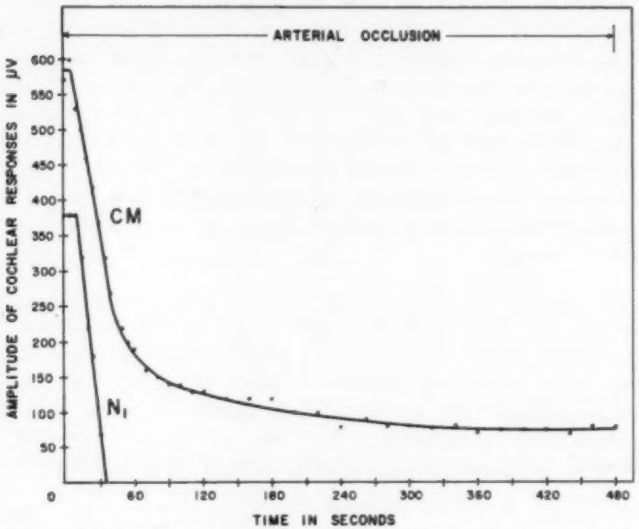

Fig. 3. Graph showing changes in Neural (N_1) and microphonic (CM) response of the cochlea after arterial obstruction.

stimulus was about 1 millisecond or less. Cochlear responses were photographed on a dual beam oscilloscope before, during, and after occlusion of the vascular supply to the inner ear. The time of each occlusion varied from one minute to 65 minutes. In 14 animals only one period of occlusion was given, and in two others the procedure was repeated two or three times.

During occlusion, the blood flow in the cochlea was moni-

tored continuously by observing the radiating arteriole through the thinned capsule in the apex.

RESULTS.

A. Survival Time.

The survival time is defined as the interval between the onset of occlusion and the instant the responses disappear. According to Gerard, it is an index of the oxidizing reserve; energy provided by other reactions, such as glycolysis, and metabolic rate.

There was an interval of about ten seconds from the beginning of occlusion to the first signs of depression in cochlear

TABLE I.

SURVIVAL TIME OF COCHLEAR RESPONSES.

Cochlea (clamping trachea)	Cochlea (arterial occlusion)	CNS (arterial occlusion)
CM .....80-120 sec.	CM .....75-85 sec.	Cerebellar gray....10-20 sec.
N_1 .....120 sec.	N_1 .....30-45 sec.	Cerebral cortex....14-15 sec.
	N_2 .....20-45 sec.	Corona radiata....20-25 sec.
		Medulla .....30-120 sec.

responses. Decay of the neural components (N_1 and N_2) seemed to precede that of the cochlear microphonics (CM) by a few seconds. The decay of all responses closely followed a linear function of time, but at different rates. Action potentials decayed faster than CM. This observation is illustrated in Fig. 3, in which the amplitude of CM and N_1 of a representative animal (see Fig. 1), is plotted as a function of time.

The survival time of cochlear responses are summarized in Table I, which also includes the survival time of cochlear responses as determined by clamping the trachea and the survival time of special areas of the central nervous system as determined by occlusion of the vascular supply.

The results clearly indicate that the sensitivity of cochlear primary neurons to oxygen deprivation is close to that of nervous tissue in the central nervous system. The generators

of the oxygen dependent portion of cochlear microphonics (hair cells of organ of Corti) are twice as resistant to oxygen lack as the generators of action potentials (primary neurons). The information does not tell whether the depression of cochlear microphonics is due to impairment of hair cells or decay of the DC resting potential in the cochlear partition. After the investigations of Bekesy[16] and Davis, et al.[17] we may assume that the depression of CM may be due to metabolic impairment of both hair cells of the organ of Corti and the stria vascularis.

The appraisal of survival time clearly indicates that the oxidative reserve within the cochlea is quite small and/or the oxygen requirements of the generators of electrical activity is rather large. Almost immediate drop in available oxygen in cerebral tissues after vascular occlusion is shown with the polarographic method.[7,18] Rapid drop in available oxygen in the cochlea produced by nitrogen breathing has also been demonstrated.[19]

The longer survival times obtained by clamping the trachea (see Table I) were due to the oxygen supplied by the circulating blood.

RECOVERY TIME.

The recovery time is defined as the interval from the end of occlusion to the first signs of recovery of responses. According to Gerard,[13] this time is an index of the speed of physico-chemical reconstruction of cells.

In our series, several animals showed no recovery of either function or circulation when the occlusion was discontinued. This suggested that the procedure of compressing the vascular supply might produce damage to both nerve fibers and blood vessels. Determination of recovery time and time required for complete recovery of cochlear responses were ascertained in those animals which showed reversal of both function and circulation. Because of uncertainties the quantitative determination will be postponed for further inevstigation. In the present report only some qualitative aspects are presented.

In general, short periods of occlusion (less than five min-

utes) were followed in a few seconds by recovery of cochlear microphonics followed by recovery of the action potentials. As time of occlusion was prolonged the recovery time of CM was little affected, while that of N_1 became longer.

Complete recovery of cochlear responses was observed after occlusions of less than eight minutes. Above 30 minutes of occlusion neither CM nor N_1 seemed to reach even 50 per cent of the reference amplitude. Although we do not have observations on occlusions between eight and 30 minutes, we may infer that severe depression of cochlear function may follow complete interruption of the vascular supply of about 30 minutes. The question whether this impairment is permanent or transitory remains to be investigated. Recent observations by Meyer on occlusion of the middle cerebral artery indicate that permanent impairment of motor function required at least 50 minutes of occlusion. If cortical oxygen tension was below 20 per cent for longer than one hour, infarction regularly occurs. Shorter periods of occlusion produced temporary paralysis that could be aggravated by reducing the systemic blood pressure. The electroencephalogram followed the changes in circulation closely, but functional recovery lagged behind restoration of blood flow, oxygen tension, and the electroencephalogram.

Another feature of recovery which needs further investigation is the supernormal response of neural components immediately following a short period of occlusion. The supernormal response of action potentials parallelled a rather large increase of the summating potential. This study may be important for the understanding of present concepts in cochlear physiology. The supernormality of electrical activity corresponds to that of supernormal blood flow recorded in the moving pictures. Meyer et al.[12] found a similar effect on the available oxygen in the cortex on release of arterial obstruction as revealed by the polarographic method, and interpreted this as reflecting both increased blood flow and reduced oxygen consumption of the nervous tissue.

Our observations indicate that the generators of cochlear microphonics possess a ready mechanism for physico-chemical

reconstruction, while that of the neural components seemed to be slow and easily damaged.

REVIVAL TIME.

The revival time is defined as the maximum duration of occlusion after which responses can return. According to Gerard,[1] it is an index of rate of occurrence of changes due to proteolysis, accumulation of metabolites and the like.

Our observations, though not yet completed, indicate that the survival time of CM is about 60-65 minutes and that of action potentials about 50 minutes.

C. Histological Findings: The ears of 50 animals were prepared for histological examination following temporary arterial obstruction. These included obstructions of 3, 5, 10, 15, 20, 30, 45, 60 and 120 minutes. Definite histological changes can be produced by temporary obstruction of the internal auditory artery. These changes are unlike those produced by permanent venous obstruction or permanent arterial obstruction. In general, the longer the obstruction the more severe the damage. Because of technical difficulties, obstruction was sometimes incomplete. This resulted in variation in the degree of cell damage within the group with similar durations of obstruction. Interruption of blood flow for only a few minutes does not produce cell changes in the cochlea. With obstruction of five minutes or more, spotty small lesions appear. The reason for this distribution of damage is not evident. The cochlear structures are much more vulnerable than the vestibular. Within the cochlea, the hair cells, cells of the limbus spiralis, and the spiral ganglion cells are the principal structures involved. Hair cell lesions may lead to spiral ganglion cell degeneration if the animal is allowed to survive for over two weeks. This type of secondary change in the spiral ganglion cells is evident in short (5-15 minutes) arterial obstructions. The independent response of these ganglion cells, however, is apparent in short term experiments before secondary degeneration can take place. Thus, in animals sacrificed within a few days, ganglion cells show changes only after arterial obstructions of 30 minutes or more. Hair cell damage is scattered throughout the cochlea but varies

from animal to animal and is not extensive even after one hour obstruction. The basal turn is perhaps more commonly affected. When the arterial supply is permanently obstructed either by trauma from the probe or direct coagulation of the vessel, disintegration of the cochlea sets in almost at once, and continues to complete necrosis in less than 24 hours. In contrast, the lesions produced by temporary arterial obstruction are very small and non-progressive. In the short obstructions (under 15 minutes) the outer hair cells are more often affected than the inner hair cells and supporting cells. With longer obstructions, both inner and outer hair cells and pillar cells are affected; however, chronic venous obstruction also produces outer hair cell damage. These findings suggest that in general the outer hair cells are more vulnerable when blood flow is impaired. In marked contrast to the findings in chronic venous obstruction atrophy of the stria and spiral ligament was minimal. Hemorrhage, spiral ligament degeneration and pigment cell migration were completely absent. Similar to the findings in chronic venous obstruction and permanent arterial obstruction, endolymphatic hydrops or collapse of the cochlear duct did not occur. The histological technique was adequate to show this pathology, and in a few instances definite labyrinth hydrops was seen in the control ear, associated with severe middle ear infection. When the vestibular part of the labyrinth was involved (obstruction of one hour or longer) the hair cells in their end-organs were most susceptible to damage. There were no changes visible in the endolymphatic sac. Since blood flow in these areas was not examined, its relation to vestibular cell damage cannot be clearly defined.

Representative changes are illustrated in Figs. 4 to 10.

Our findings of limited histological damage in the inner ear after one hour of arterial obstruction may be compared with those of Meyer.[20] He reports that infarction of the cortex occurs when the local oxygen tension falls below 20 per cent for more than one hour.

The recognition of significant histological changes in the labyrinth following temporary obstruction of the arterial

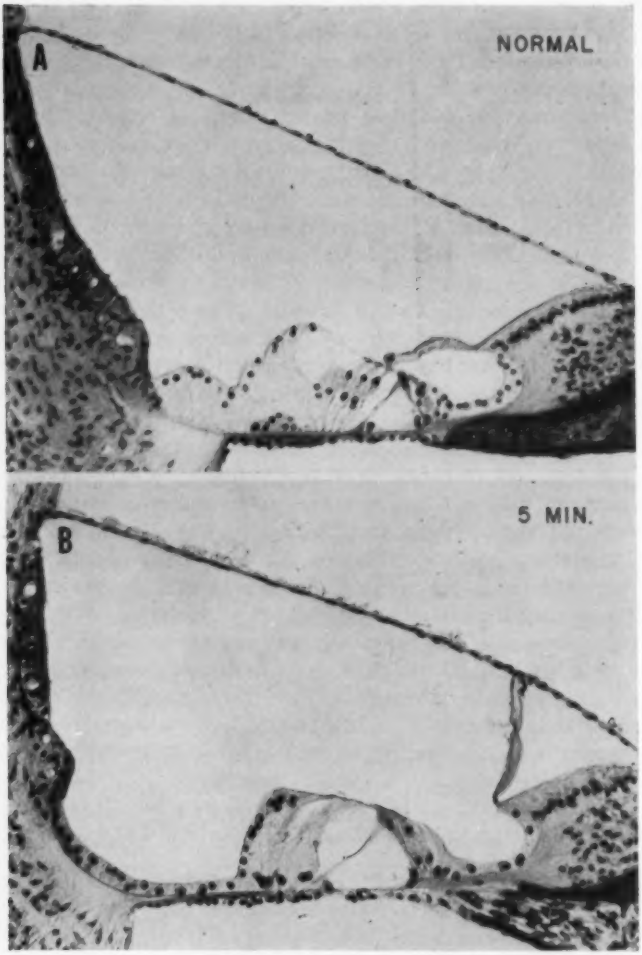

Fig. 4. a.—Normal cochlear duct. b.—Loss of outer hair cells 45 days after five minutes arterial occlusion.

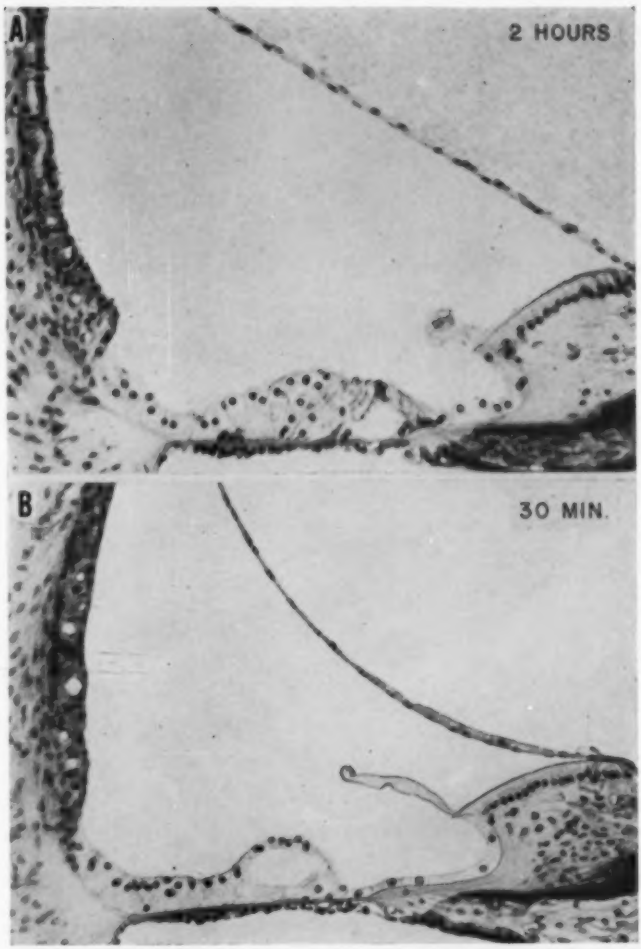

Fig. 5. a.—Loss of outer and inner hair cells and limbus cells four days after two hours of arterial obstruction. b.—Loss of sensory and supporting cells with normal stria three months after 30 minutes of arterial obstruction.

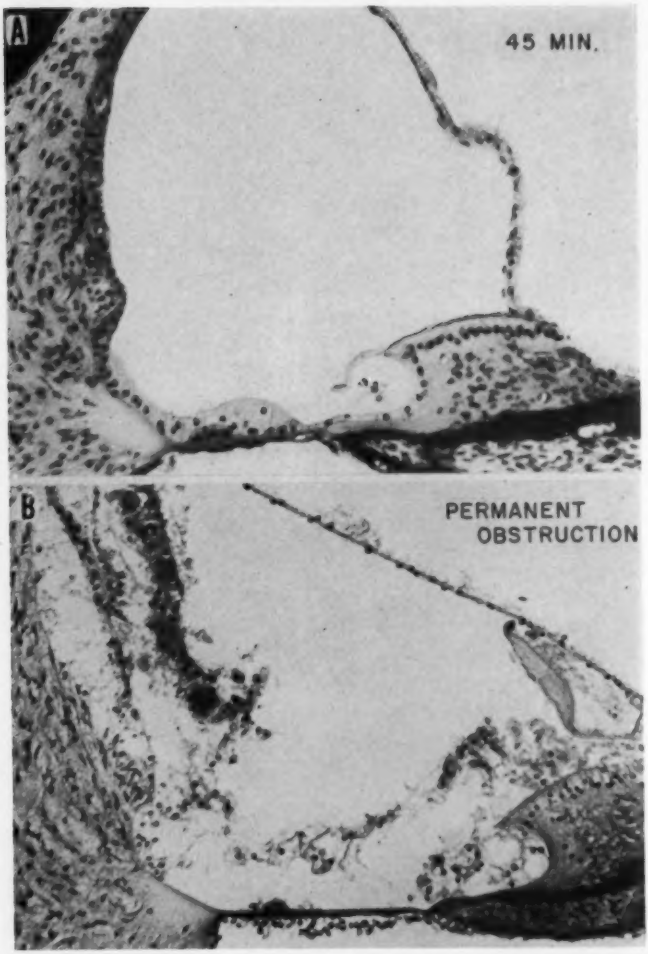

Fig. 6. a.—Loss of organ of Corti and atrophic stria one month after 45 minutes arterial obstruction. b.—Necrosis of stria and end-organ one day after complete and irreversible obstruction.

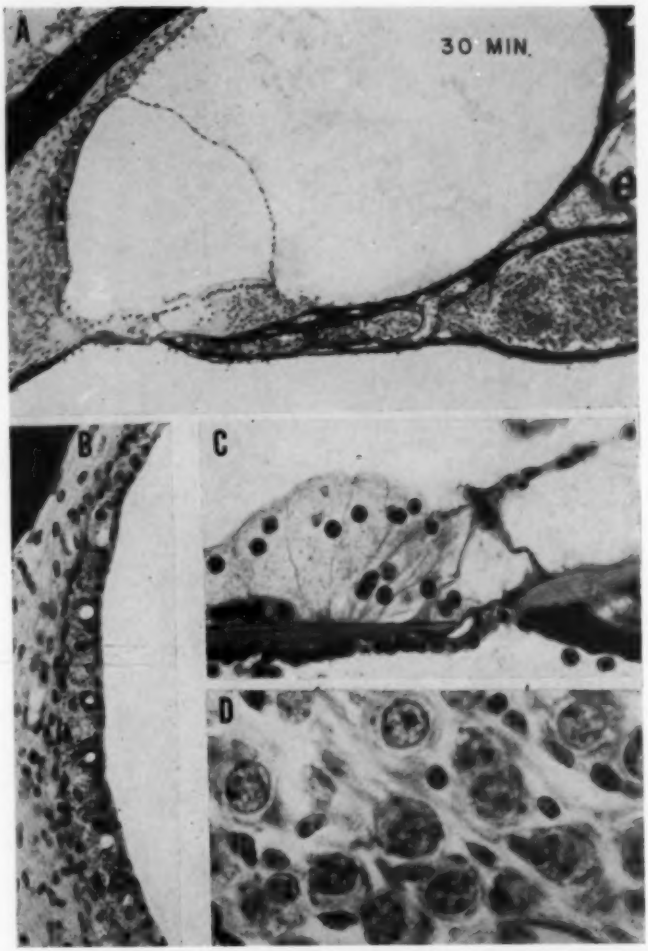

Fig. 7. a.—Inner and outer hair cell damage one day after 30 minutes arterial obstruction (higher power in b, c and d). b.—Stria unaffected by this temporary obstruction. c.—Hair cells in organ of Corti seen in a. d.—Adjacent spiral ganglion cells show minimal changes.

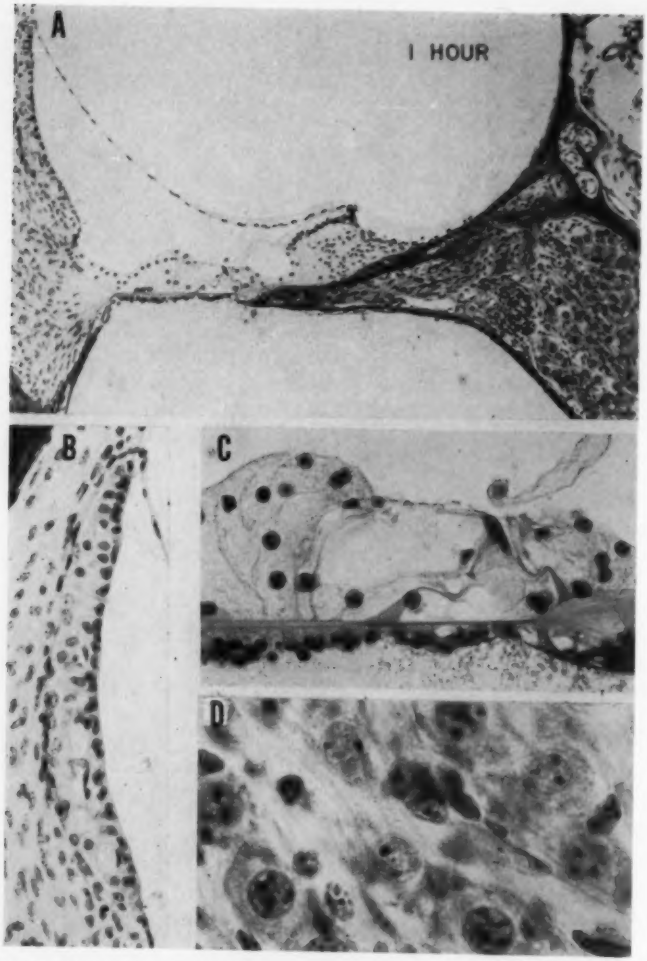

Fig. 8. a.—Sensory cell and spiral ganglion cell damage one day after one hour arterial obstruction (higher magnifications in b, c, d). b.—Minimal edema of the stria vascularis. c.—Details of hair cell damage. d.—Early spiral ganglion cell changes.

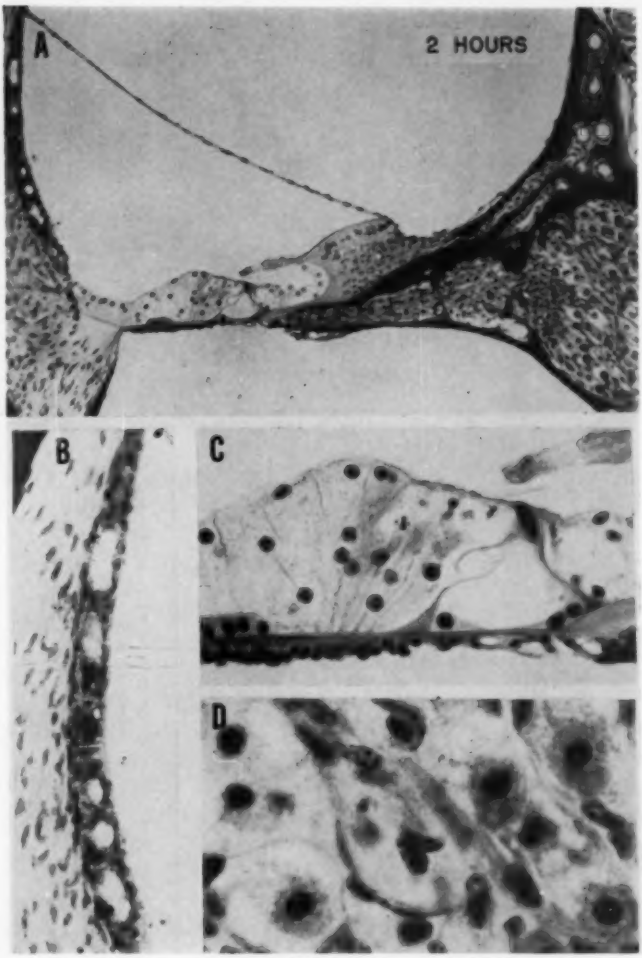

Fig. 9. a.—Hair cell, spiral ganglion and strial changes one day after two hours of arterial obstruction (higher magnifications in b, c, and d). b.—Vascular changes in stria. c.—Sensory cell damage in organ of Corti. d.—More advanced ganglion cell lesion than in Fig. 6.

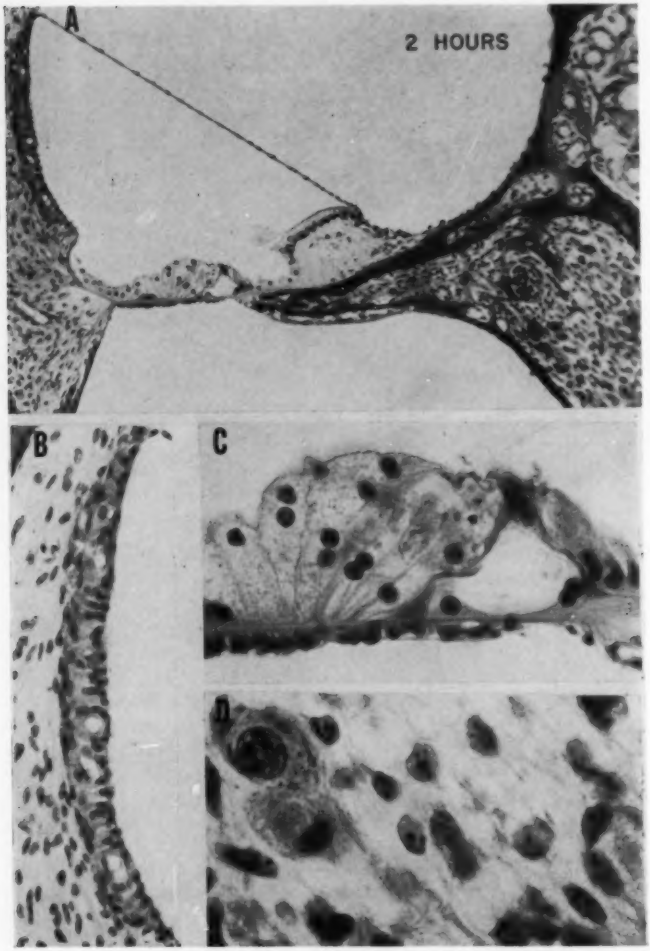

Fig. 10. a.—Hair cell, limbus and ganglion cell damage four days after two hours of arterial obstruction (higher power in b, c, and d). b.—Normal stria. c.—Inner and outer hair cell lesion. d.—Dissolution of spiral ganglion cells.

supply was aided by the presence of the unoperated ear on the same slide for comparison; furthermore, previous studies on permanent venous and arterial obstruction of the labyrinth revealed characteristic histological changes. Rapid necrosis of the labyrinth seen in sections from permanent arterial obstruction was in marked contrast to smaller lesions more slowly developing and associated with labyrinth hemorrhage, seen in chronic venous obstruction. This permitted the separation of unsatisfactory experiments where either or both of the above vascular lesions resulted from the surgical procedure. Because of the surgical approach, permanent damage to the venous outflow or to the arterial supply to the labyrinth occasionally resulted. This was usually recognized at the time of operation by observing changes in blood flow and diameter of the radiating arteriole through the capsule bone, and fitted in with these characteristic histological changes subsequently found.

Another problem that arose in the histological evaluation of this vascular lesion, was the unexpected trauma to the VIIIth nerve trunk that occasionally followed manipulation with the probe. In this series of experiments it was possible to find a group with uncomplicated nerve damage that showed degenerative changes extending through the spiral ganglion to the end-organ typical of nerve trauma. These degenerative changes were apparent only when the animal was kept alive at least two weeks. For this resaon, animals that were sacrificed before this period of time had elapsed and with various durations of arterial obstruction, were the most satisfactory in evaluating the significant changes following the vascular lesion.

GENERAL SUMMARY.

It is possible to interrupt the arterial supply to the inner ear by pressure with a probe over the internal auditory artery. On release of pressure, flow returns to normal after first going through a supernormal period. Blood flow may return to normal even after one hour of obstruction without visible permanent change in the blood vessels or their surroundings. The short period of supernormal flow is probably due to tem-

porary dilation of the internal auditory artery and its large tributaries on the basis of trauma and local anoxia.

The cochlear function as reflected in microphonic and action potentials is profoundly depressed within 60 seconds after complete interruption of the arterial supply but may return to normal even after eight minutes of complete obstruction. If blood flow stops for over 30 minutes, there is only partial recovery of electrical responses.

Histological changes in the labyrinth are produced by temporary arterial obstruction. The external hair cells and the ganglion cells of the cochlea are particularly vulnerable, while the vestibular end-organs are least affected. Even one hour of obstruction produces minimal changes throughout the cochlea. These changes are unlike those produced by permanent obstruction of the internal auditory artery or permanent obstruction of the inferior cochlear vein and its tributaries. Endolymphatic hydrops does not follow temporary obstruction of the internal auditory artery.

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COURSE IN AUDIOLOGY AND OTOLOGY.

The program of the II Audiology and Otology Course which the Departmento of O.R.L. of Hospital San Jose of Bogota, Colombia, is organizing for the month of August, 1959 includes the names of many otorhinolaryngologists of Colombia, Venezuela and Ecuador. The price of this course is $1,000 and is limited to 20 matriculants. The course in audiology only is $500.00. The closing date for registration is July 10, 1959. For further details write Jorge Garcia Gomez, Head of the E.N.T. Department Hospital, San Jose, Bogota, Colombia.

TYMPANOPLASTY.*†

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INTRODUCTION.

The radical mastoid operation as commonly performed in the past often resulted in further hearing loss and, therefore, created among both otologist and patient a dislike for this operation. Consequently, many surgeons refused to "clean out" the tympanic cavity and so were able to preserve a practical hearing level in some cases. These conservative operations consisted of removing disease from the attic, antrum or mastoid and sometimes from the mesotympanum while preserving mucosa, ossicles and any remaining tympanic membrane. An anatomical mechanism providing a sound pressure difference at the windows was sometimes established, quite inadvertently, by these methods. This conservative approach often failed, however, because of recurrence of cholesteatoma and continued suppuration.

With the aid of antibiotics and the operating microscope, associated with a better understanding of skin grafting techniques and middle ear physiology, surgery for the correction of suppurative middle ear disease can be performed with a better opportunity of inactivating the disease while preserving or improving the hearing. We are indebted, of course, to Wullstein[1] and Zollner[2] for providing us with refinements in tympanoplastic technique.

PREOPERATIVE EXAMINATION.

The preoperative examination must assess the extent of

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disease in the middle ear, mastoid, attic and eustachian tube regions and provide a quantitative evaluation of auditory function. The ear should be thoroughly cleaned with a small suction tip, inspected, and possibly probed under visual magnification. An attempt should be made to evaluate the functional status of the ossicles and ear drum and the condition of the middle ear mucosa. Inspection under magnification should determine the presence and extent of squamous cell

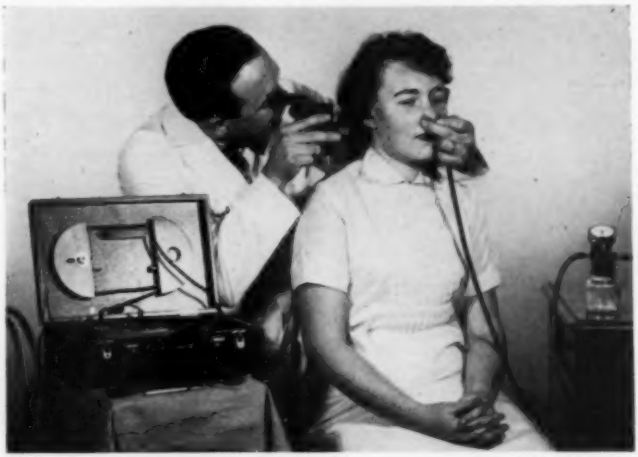

Fig. 1. The combined use of controlled inflation and inspection under magnification is essential for preoperative examination and for postoperative evaluation. The Hallpike microscope used in conjunction with the Senturia inflator has been satisfactory in our experience.

epithelium in the middle ear and epithelialized tracts extending into the antrum and attic. The eustachian tube can be tested for patency by the Valsalva technique using controlled air pressure (from 30 to 80 mm. mercury) while inspecting the middle ear with magnification (see Fig. 1); thus, perforations and false membranes can be studied and hidden purulent material can be detected. The sound probe test is useful in evaluating the functional intregrity of the ossicular chain.[3] The crossed stapedial reflex may be helpful in determining the mobility of the stapes. Roentgenograms of the

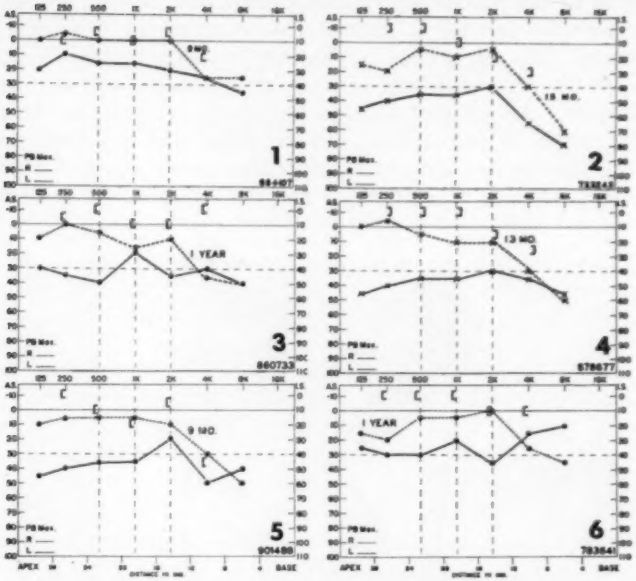

Fig. 2. Examples of successful Type I tympanoplasties. We have plotted the frequencies on the abscissas of the audiograms in accordance with their approximate spacial distribution along the Cochlear duct; thus, the low frequencies are somewhat crowded at the left end of the scale. Distance along the Cochlear duct is indicated in millimeters at the bottom of the charts. The ordinate on the left shows hearing loss in decibels by the American Standard and on the right by the International Standard, the latter corresponding closely to the current British Standard. The American Standard is based on a U. S. Public Health Service population survey and is based on average normal hearing whereas the International (or British) Standard is based on the median value for the hearing of well-motivated young adults, otologically normal and carefully tested.

temporal bone may reveal gross changes but are of little help in revealing pathological changes which are of technical importance in tympanoplasty.

Auditory tests should establish not only the pure tone thresholds for bone and air but the discrimination score as well. If the predicted value of tympanoplasty surgery is insignificant (*e.g.*, severe bone conduction loss in the presence of a good ear on the opposite side), a radical mastoidectomy may be done. During the period of this report, we performed radical mastoidectomy for chronic suppuration on two ears in which

preoperative tests revealed profound sensory-neural type deafness.

TYPES OF TYMPANOPLASTY.

Tympanoplasty has a dual objective: the first is elimination of disease, and the second is reconstruction of the sound transmitting system. Reconstruction is attempted only after

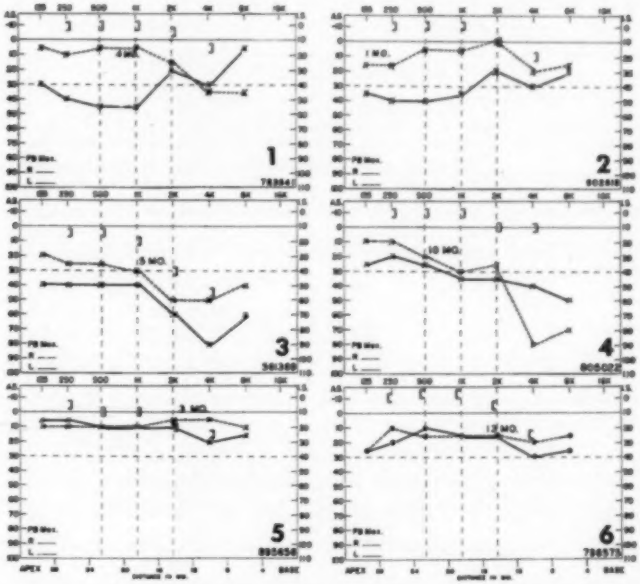

Fig. 3. Examples of successful Type II-A tympanoplasties. Cases 2, 3, 4 and 5 without mastoidectomies.

the underlying disease has been completely irradicated. The surgical methods which will eliminate disease while preserving middle ear mucosa and the conducting mechanism are technically difficult and the end results are largely dependent upon the skill and experience of the surgeon.

The theory of tympanoplasty has been discussed in detail

elsewhere.[4] In brief, the objective is to create a sound pressure differential between the oval and round windows (with or without the sound conducting mechanism).

The meticulous classification of tympanoplasty operations permits one to predict the patient's chances for improved hearing and allows the surgeon to compare his results with

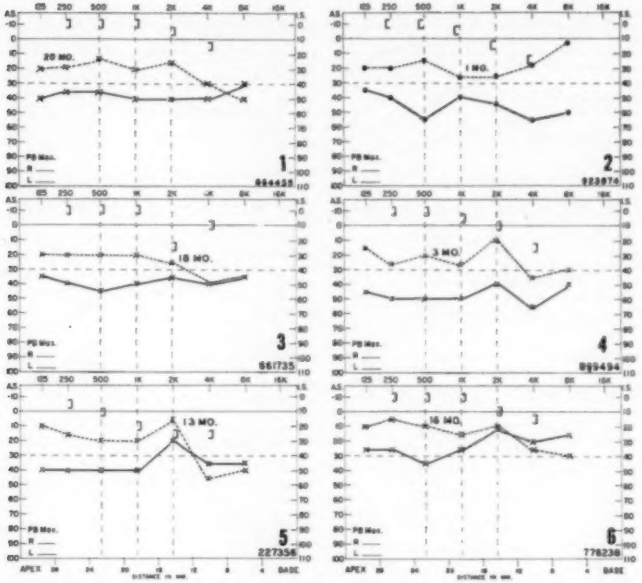

Fig. 4. Examples of successful Type III-A tympanoplasties. Case 2 without mastoidectomy.

those of others. We have followed Zollner's modification[5] of the classical Wullstein classification (see Table I).

Type I: Plastic Repair of Drum with or without Meatoantrotomy.

This operation consists of the plastic repair of a perforation of the tympanic membrane and it may include reflecting the

ear drum forward and removing some of the posterior bony annulus so as to inspect the ossicles, windows, sinus tympani and antrum. The existing pars tensa and annulus fibrosus are preserved and denuded of epithelium along with the adjacent canal wall (if necessary) to provide an adequate bed for the graft. We almost always use the post-auricular approach to the tympanic space. After elevating a meatal skin flap, the external auditory canal is widened with a bur (except in those patients having large canals or those with small easily visible perforations). The full-thickness skin graft*

TABLE I.

Types (Zollner).
I—Plastic repair of drum with or without meato-antrotomy.
II-A—Attico-antrotomy with plastic repair of drum.
II-B—Simple attico-antrotomy.
III-A and IV-A—Total plastic repair (malleus and incus destroyed). Large graft.
III-B and IV-B—Minor plastic repair (malleus and incus destroyed), half or more of drum remaining. Graft used as necessary to complete closure.
V—Plastic operation and subsequent fenestration.

(III—placed on stapes.) (IV—placed on promontory.)

bridges the tympanic space and extends onto the posterior canal wall as necessary to cover bone exposed as a result of enlarging the ear canal. We occasionally permit large grafts to lie on a small de-epithelialized area on the promontory to assure nourishment of the central part of the graft.

Type II-A: Attico-antrotomy with Plastic Repair of Drum.

Plastic repair of the tympanic membrane is accomplished with or without meato-antrotomy plus exposure and exenteration of disease in the attic. The skin graft may extend onto the head of the malleus and body of the incus, bridging to the canal wall or into the mastoid cavity if a mastoid exenteration is done. If the operation is done without a mastoidectomy, it is essential that a mucosal-lined tract extend from the anterior tympanic region through the superior aspect of the attic

*In reality, these grafts, although removed as full-thickness, are thinned with scissors to the junction of the papillary and reticular layers of the derma.

to the mastoid so as to allow aeration of the mastoid cells. It is often important to determine the condition of the mastoid antrum by either a limited meato-antrotomy or a superior observation window. The observation window is made in the posterior-superior canal wall and allows inspection of the mastoid antrum and posterior part of the epitympanic space

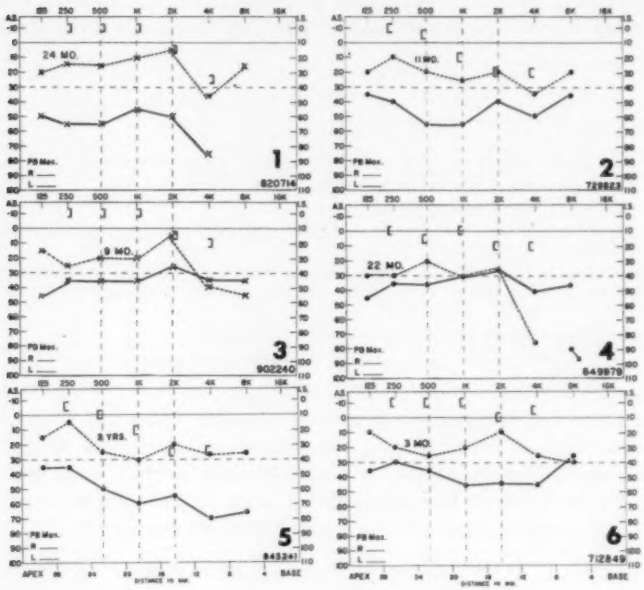

Fig. 5. Examples of successful Type IV tympanoplasties. Case 2 without mastoidectomy.

and is covered by the skin graft. Even though a mastoidectomy is necessary, it is desirable to preserve bony annulus when possible, for this "bridge" minimizes cicatricial immobilization of the incus, especially if the mucosa is preserved in this area. Included in Group II-A are patients in whom the incus or malleus or both have been transposed between the graft and the stapes creating thereby a "columnella" effect.

Type II-B: Simple Attico-antrotomy.

This operation is a well-known type of modified radical mastoidectomy. The pars tensa and ossicular chain are intact, and the operation is directed at meticulous removal of disease from the attic region. The mastoid may be exenterated and if so, an attempt is made to preserve the "bridge." In such cases, the skin graft passes from the upper part of the tympanic membrane to the "bridge," thereby closing the attic perforation and extends into the mastoid cavity. Again, if the mastoid is not diseased, it is not necessary to perform a mastoidectomy as long as a mucosal-lined tract extends from the anterior tympanic region through the epitympanum to the mastoid air cell system to assure aeration of the mastoid.

Types III-A and IV-A: Total Plastic Repair (drum, malleus and incus destroyed); Large Graft.

Wullstein has classified those operations in which the ear drum or graft is placed upon the head of the stapes as Type III and those in which the oval window is exteriorized and the graft or tympanic membrane is laid upon the promontory as Type IV. This classification takes into account the physiological differences occurring when sound is transmitted to the head of the stapes through a membrane and that occurring when sound strikes the footplate directly.

Zollner has subclassified these types into the "total plastic" (Type III-A and IV-A) and the "minor plastic" (Type III-B and IV-B). In the total plastic, there is complete loss of the tympanic membrane requiring that the graft bridge the gap from the annulus to either the stapes (Type III-A) or promontory (Type IV-A). In the minor plastic there is preservation of half or more of the ear drum and the graft is used if necessary to complete the closure, either to the stapes (Type III-B) or to the promontory (Type IV-B). We utilize Zollner's subclassification because it seems that a good result depends more upon the condition of the mucosa and the size of the graft than it does upon whether or not the reconstruction utilizes the "columnella effect" of the stapes.

Diseased tissue is removed meticulously from the tympanic space using great care not to damage the mucosa. These

operations can be performed with or without mastoidectomy. The graft lays on the de-epithelialized antero-inferior canal wall and bridges the tympanic space to the promontory or head of the stapes, whichever is appropriate.

Types III-B and IV-B: Minor Plastic Repair (malleus and incus destroyed); Remaining Drum Used with Small Graft.

When half or more of the tympanic membrane remains, there is often healthy mucosa in the hypotympanum. The success rate is much higher than in the total plastic regardless of whether the graft or tympanic membrane attaches to the head of the stapes or to the promontory. If the existing tympanic membrane is inadequate in itself then a graft is used to complete the closure; again, the operation may be done with

TABLE II.

MASTOIDECTOMY.

Type	Total Number	With Mastoid	Without Mastoid
II-A and II-B	17	6	11
III-A and IV-A	82	71	11
III-B and IV-B	20	16	4
Total	119	93 (78%)	26 (22%)

or without a mastoidectomy. A radical mastoid cavity should not be created unless necessary, for to do so creates a post-operative surgical state which occasionally requires interval care for many years. In our series of 119 patients of Types II, III and IV, mastoidectomy was avoided in 26 (22 per cent). (See Table II.)

In all Type III operations, it is essential that the facial ridge be de-epithelialized, for the membrane (drum or graft) must contact this region to maintain its position on the head of the stapes. In Type IV tympanoplasty, it is not known whether it is best to preserve the mucosa on the footplate or remove it, and if the mucosa is removed, whether the footplate should be left denuded or covered with a graft. We have employed all these methods with success.

Type V: Plastic Operation and Subsequent Fenestration.

This procedure applies only to ears with fixation of the stapedial footplate and must be done in two stages. The first objective is to create both a mastoid cavity and a sealed-off hypotympanic (and possibly mesotympanic) space encompassing the round window. The adequacy of this tympanic

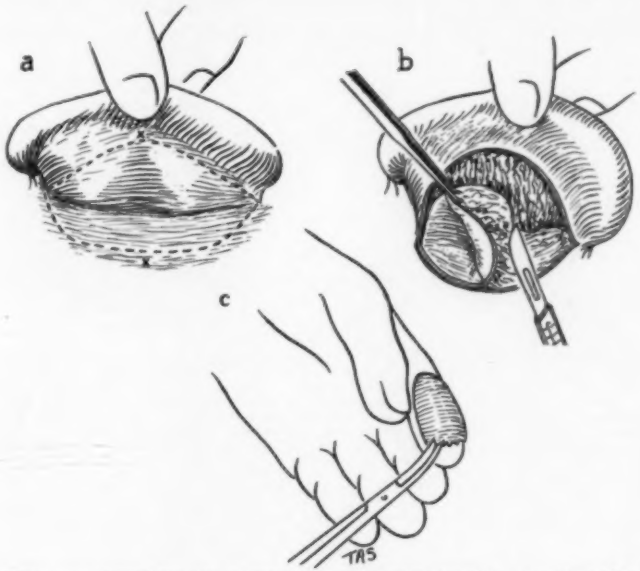

Fig.6. a.—Incisions for removal of post-auricular skin. b.—The skin is removed with a sharp knife, cutting through the underlying fatty subepithelial layers. c.—The skin is thinned with a sharp curved scissors, either on the finger or on a block. When the skin graft is to lie on the antero-inferior canal wall folding of the graft can be minimized by serrating this margin.

space should be established with certainty by controlled inflation and simultaneous inspection of the graft under magnification before the second stage (fenestration operation) is performed.

FURTHER COMMENTS ON TECHNIQUE.

The post-auricular skin contains little elastic tissue and,

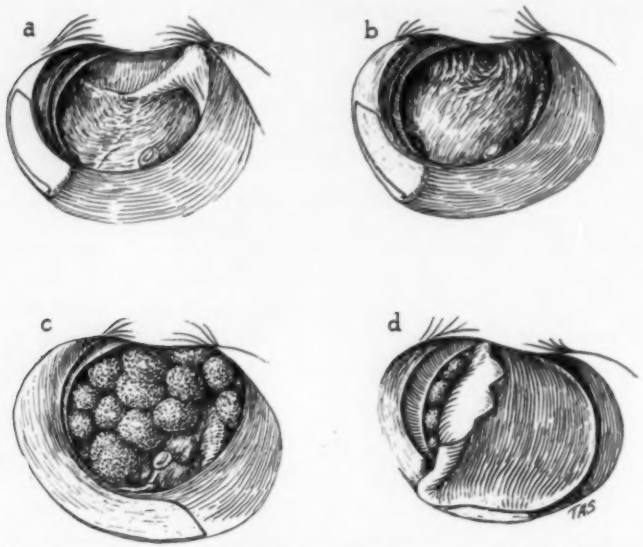

Fig. 7. Technique for Type III-A tympanoplasty without mastoidectomy. a.—The tympanic membrane and the incus are missing. The malleus is present and the handle is adherent to the promontory. The external auditory canal has been enlarged and the skin of the posterior canal has been reflected inferiorly. b.—The malleus has been removed and the attic and antrum have been inspected and found to contain healthy mucosa. c.—The antero-inferior canal wall has been de-epithelialized, the facial ridge has been denuded and the tympanic space and attic filled with small pieces of gelfoam. The meatal flap has been replaced against the posterior canal wall. d.—The full-thickness skin graft is placed so as to lie on the antero-inferior canal wall and extend across the tympanic space to the head of the stapes and facial ridge and back to the posterior canal wall. The graft is fitted onto the posterior canal wall so as to cover any bare bone.

therefore, when used as a full-thickness graft, it is easier to manipulate and maintain in position than split thickness skin. Our post-auricular grafts vary in size from 2 to 3 cm. in width to 4 to 6 cm. in length. We remove the skin at the beginning of the operation and keep it in heparinized refrigerated blood until time for introduction. It is then thinned with a small curved scissors and trimmed to proper size. The tympanic space is filled with pieces of gelfoam to aid in supporting the graft. The question as to whether gelfoam stimulates connective tissue proliferation has not been completely answered. Grafts which extend upon the antero-inferior canal wall are serrated at its margin to facilitate introduction

without the formation of folds (see Fig. 6-c). The graft is meticulously approximated to its bed, trimming as necessary to avoid folds. Small cotton balls soaked in Ringer's solution are used to hold the graft tightly into its bed, particularly in the acute angle at the annulus antero-inferiorly. An attempt is made to stretch the graft across the tympanic cavity under some tension. When the graft is to cover the footplate, it is invaginated into the oval window with a small cotton ball. The entire cavity is lined with rayon strips, after which larger

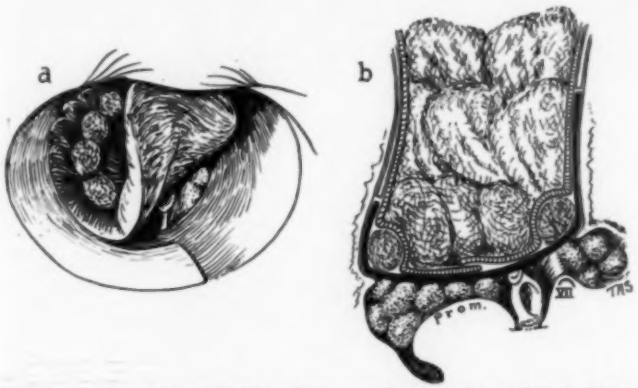

Fig. 8. Packing the graft into place. a.—Cotton balls soaked in Ringer's solution are used to hold the graft against the canal wall at the annulus as necessary. b.—Rayon strips are used to cover the entire cavity so as to create a complete sleeve. Larger cotton balls soaked in Ringer's solution are introduced into the bottom of the sleeve and packed firmly against the canal walls. Further packing consists of synthetic sponge rubber and vaseline gauze.

cotton balls are appropriately placed so as to create firm pressure against the graft. This is followed by pieces of synthetic rubber and finally vaseline gauze packing (see Figs. 6, 7, 8). Perre[6] stresses the importance of accurate approximation and firm pressure and we are inclined to agree with him. Split thickness grafts were taken from the chest, thigh or inner surface of the arm in 14 of our series of 160 tympanoplasties. In most of these patients, full-thickness skin was not available because of post-auricular scars. These grafts were cut .015 inches (.38 mm) thick and although the skin was more difficult to handle, the results were as good

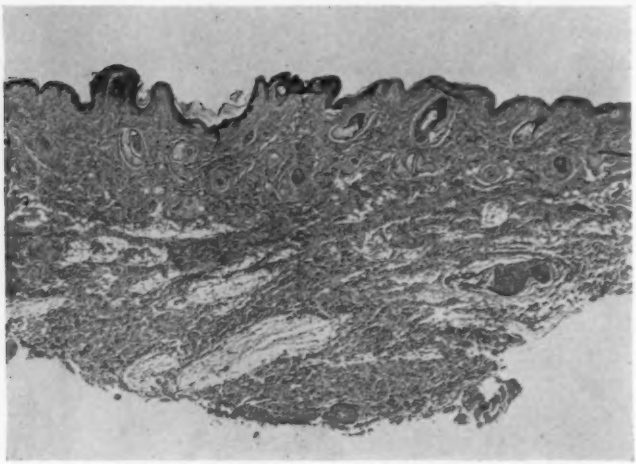

Fig. 9-a. Photomicrograph of full-thickness post-auricular skin removed at the time of surgery showing sebaceous glands and a rather thick layer of sub-epithelial fatty tissue.

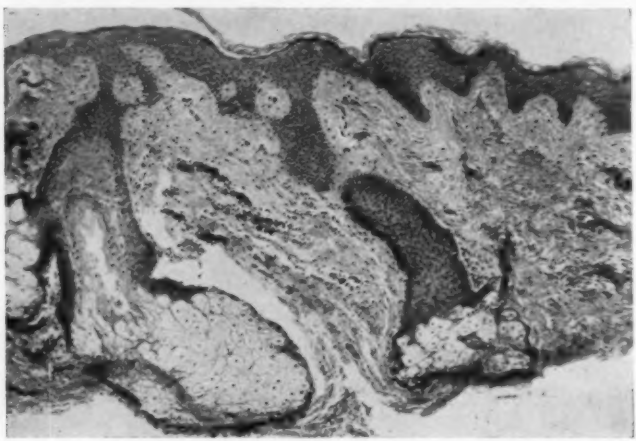

Fig. 9-b. Appearance of graft after thinning. Note the presence of sebaceous glands. The dermis contains blood vessels of the subpapillary network, the importance of which has been emphasized by Guilford and Wright[16].

as with full thickness grafts. Photomicrographs of full-thickness and split thickness grafts are shown in Figs. 9 and 10. Fig. 11 shows a full thickness graft removed six months after surgery because of a perforation. The tympanic surface is lined by a transitional epithelium.

Surgical rayon is placed upon the donor site of split-thickness grafts and covered with a gauze dressing. The following day, the dressing including the surgical rayon is removed and

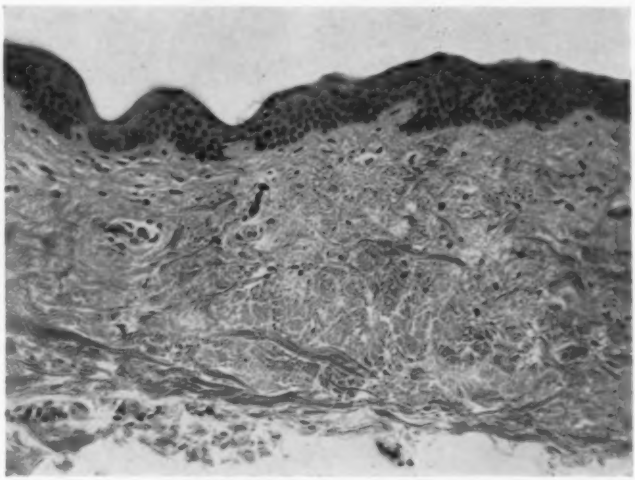

Fig. 10. Photomicrograph of split thickness skin from the anterior chest wall cut at a thickness of .015 inches. Note the absence of glands. It should be understood that the grafts appear considerably thicker in these views than they are when cut because of fibrous tissue shrinking and because of the thickening which occurs from fixation.

the donor site is exposed to the drying effect of a heat lamp for two hours morning and afternoon. Drying of the donor site permits rapid healing without crusting or adherent dressings.

The packing is removed from the ear on the seventh postoperative day, and the middle ear is inflated by the Valsalva technique using not more than 60 mm of mercury pressure (see Fig. 1).

FINDINGS AT OPERATION.

Labyrinthine fistulas were present in six of the 160 ears. In five ears, the fistulas were located in the horizontal semicircular canal and in one, there were three fistulas in the cochlear wall.

Tympanosclerosis was present in five ears in our series, and in four of these was located in the submucosal layers of

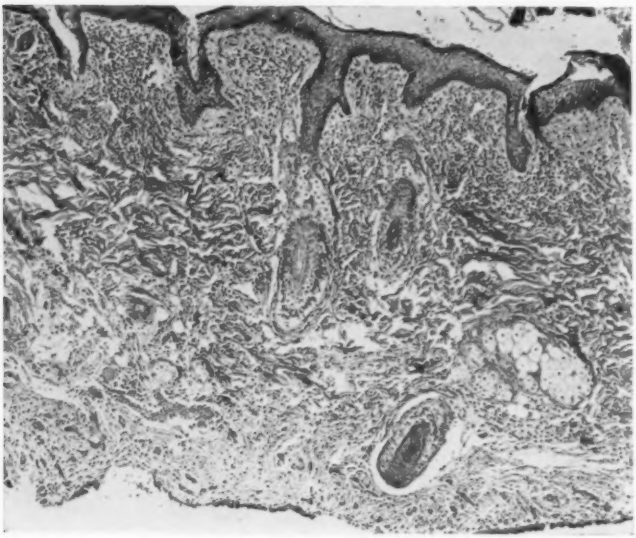

Fig. 11-a. Photomicrograph of full-thickness skin graft removed after six months because of a perforation. On the external surface, there is a normal layer of squamous cell epithelium whereas the tympanic surface is lined with a transitional cell epithelium. There are active sebaceous glands throughout the graft substance.

the tympanic cavity involving the promontory, sinus tympani, oval window and stapes, facial ridge and epitympanum. In one chronically discharging ear, there was a large ball of the material lying free in the tympanic space. In each case, it was removed with care to avoid unnecessary damage to mucosa and a satisfactory hearing status was acquired in three of the five patients.

In this condition, the mucosa of the middle ear loses its soft pinkish moist appearance and is replaced by a nodular grayish yellow or whitish hard mass. The ear may be dry with no active discharge, although there is always a history of previous long standing ear infection. The lesion usually involves part or all of the medial wall of the middle ear and may cover the promontory, round window, hypotympanum, sinus tympani and reach into the epitympanum. All of the ossicles may be

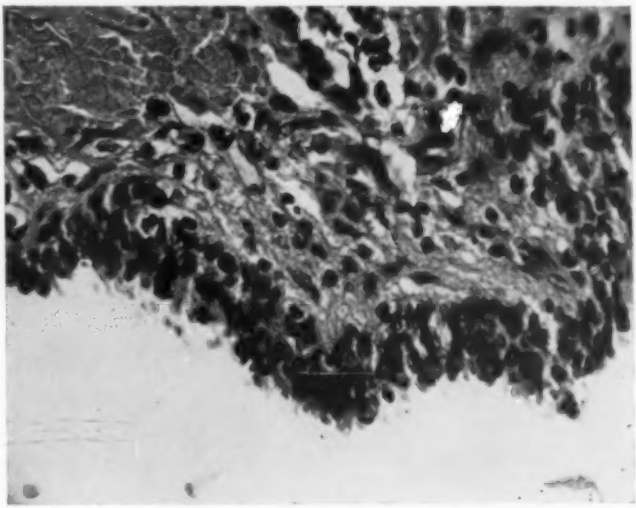

Fig. 11-b. High power view of transitional cell epithelium lining the tympanic surface of the graft.

bound down in this mass. Tympanosclerosis may not be detected preoperatively if only a small perforation is present. Zollner states that tympanosclerotic material is located in the deep layer of the submucosa and is not in direct contact with the bone of the osseous labyrinth or with the ossicles.[7]

The microscopic picture is that of diffuse hyalinization of collagenous fibers with a few scattered plasma cells and lymphocytes. There is a minimal deposit of minute calcium particles on the surface of the material (see Fig. 13).

Fixation of the stapes with otosclerotic bone was found in four patients—two in whom the diagnosis was made preoperatively. Stapes mobilization was performed at the time of the tympanoplasty, but in none was there significant postoperative hearing gain. One of these was subsequently fenestrated to become a successful Type V tympanoplasty (see Fig. 14).

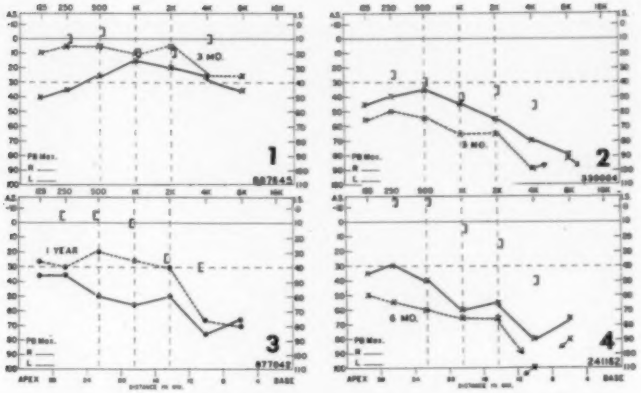

Fig. 12. Results in ears having tympanosclerosis. (1) Type I, tympanosclerosis involving crura, round window, facial ridge and hypotympanum. (2) Type II-A without mastoidectomy, large free mass of tympanosclerotic material in middle ear which could only be removed piecemeal. End-result is satisfactory even though auditory thresholds are worse. (3) Type II-A with mastoidectomy, tympanosclerosis involving all ossicles, round window niche and sinus tympani. (4) Severe tympanosclerosis involving oval and round windows, promontory, epitympanum and sinus tympani. Squamous epithelium throughout hypotympanum. Mastoidectomy performed without attempt to graft. Not included in tympanoplasty series.

Type III tympanoplasty was often necessary when there was necrosis of the long process of the incus (see Fig. 15).

In four ears with limited disease, it was possible to transpose the malleus and incus so as to establish a "columnella effect." Excellent results were acquired in three of the patients—a fourth failing because of a graft necrosis (see Fig. 16).

On seven patients, the surgery was performed when there existed a profound deafness in the opposite ear. In two of the

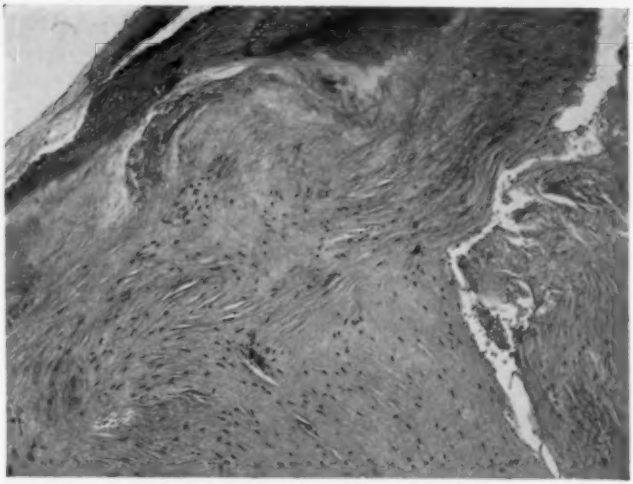

Fig. 12-a. Photomicrograph showing tympanosclerotic material removed from the promontory at the time of surgery. It consists of dense hyalinized collagenous connective tissue with minimal cellular infiltrate of plasma cells and lymphocytes.

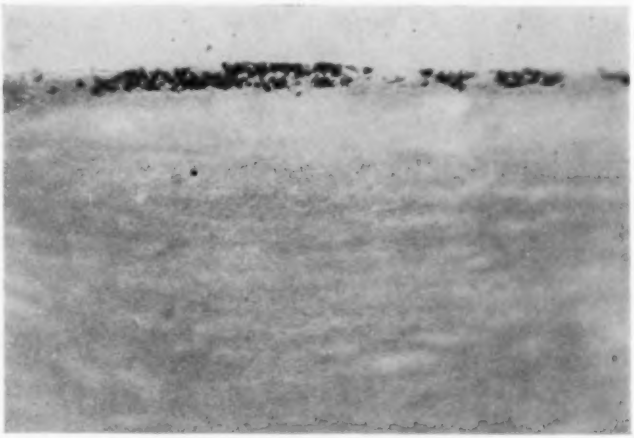

Fig. 13-b. VonKossa stain showing calcium deposit at the surface of the tympanosclerotic mass.

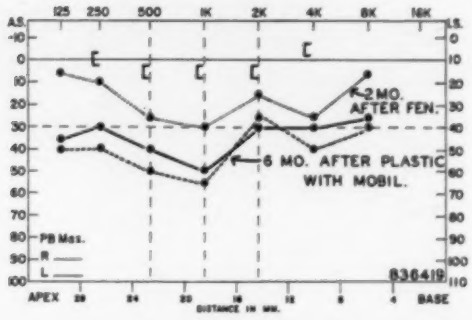

Fig. 14. Audiogram of our only Type V tympanoplasty.

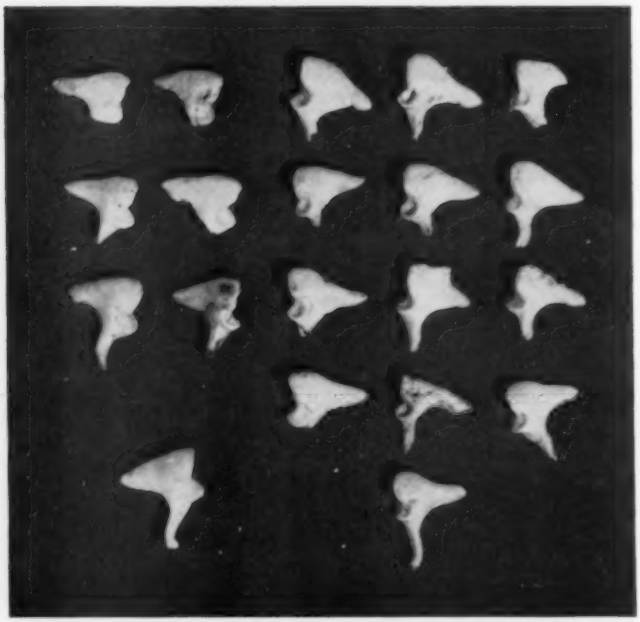

Fig. 15. Incuses removed on Type III tympanoplasty cases showing the common pathological findings of necrosis of its long process. Normal right and left incuses shown below.

operated ears, there had been a progressive drop in bone conduction thresholds during the months immediately preceding surgery. In operating these patients, great care was used to avoid opening the labyrinth. For this reason, we believe that mucosa or squamous epithelium located in either the oval or round windows usually should not be disturbed in these cases. In none of these seven patients was the hearing made worse and in four there were significant hearing gains (see Fig. 17).

It is well-known that auditory thresholds can be maintained at a functionally satisfactory level by cholesteatoma, granulations, tympanosclerosis, pus and false membranes. The cor-

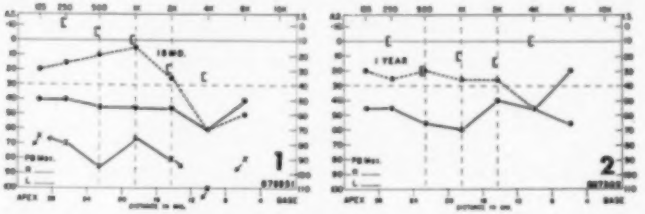

Fig. 16. Ossicular transpositions were performed in four ears with excellent results in all. Two audiograms are shown. In Case 1, an atticoantrotomy because of incudal necrosis was performed without mastoidectomy. The malleus was transposed so that the neck was interposed between the head of the stapes and the full-thickness graft. Case 2 had a severe auditory deficit in the opposite ear. These cases were classified as Type II-A tympanoplasties.

rection of these diseased states can lead to slightly worse hearing even though technically successful reconstructive surgery has been accomplished. In assessing the results of tympanoplasty, it is of importance to understand that in some cases the failure to improve hearing or even a slight aggravation of the hearing loss may constitute an excellent functional result. It is in reality, the final status of the bone-air gap which is the significant criterion of success. For example, some patients already have "Type IV" hearing because of a false membrane which has created an air pocket encompassing the round window. During the period of this report, we encountered four ears with functioning false membranes. They were preserved in two and replaced by graft in two with good results in all. If a false membrane is creating "Type IV"

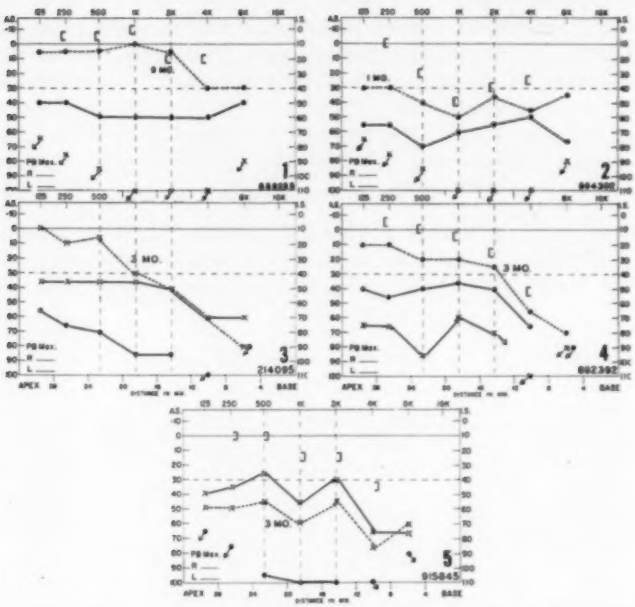

Fig. 17. Audiograms of patients having tympanoplasties in the presence of severe auditory deficits in the opposite ear. (1) III-B with mastoidectomy; (2) III-A with mastoidectomy; (3) III-A with mastoidectomy; (4) II-A without mastoidectomy.

hearing and it can be replaced by a graft so as to convert it to a "Type III" hearing, this probably should be done (see Fig. 18). We did not include in our tympanoplasty series those two ears in which false membranes were left in place.

RESULTS.

Our experience consists of 160 tympanoplasties performed during a two-year period (see Table III). The audiograms were performed in *acoustically-treated* rooms with an ambient noise level of less than 50 db in the critical frequency bands with commercially available clinical audiometers. The calibration of each audiometer was standardized regularly with the Allison calibration unit, Model 3A. Speech tests were

administered either with the above equipment or, using the regularly calibrated speech audiomter, in a two-room testing unit, using monitored live voice techniques.*

Preoperative and postoperative hearing levels were determined by averaging the three speech frequencies of 500, 1,000 and 2,000 cps. All preoperative and postoperative tests were

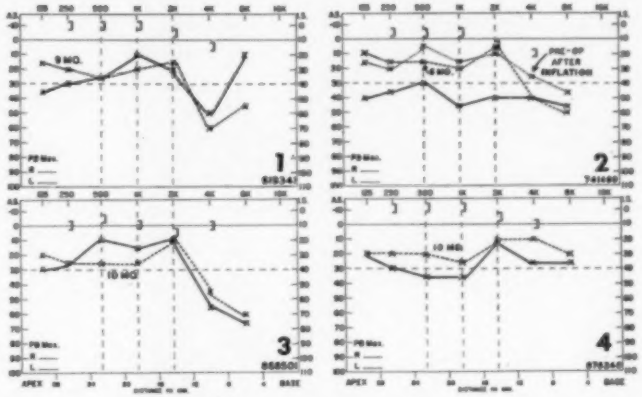

Fig. 18. Audiograms of patients having functioning false membranes prior to surgery. (1) False membrane removed and replaced by full-thickness graft, Type III-A without mastoidectomy. (2) False membrane functionally effective for few hours after inflation. Replaced by full-thickness skin graft, Type III-A with mastoidectomy. (3 and 4) False membranes creating functionally effective cavum minors. Disease removed from attic, antrum and mastoid and false membrane retained. Not included in our tympanoplasty series.

performed after inflation and careful cleaning of the ear canal, mastoid and tympanic areas.

We are aware that the Otosclerosis Study Group recently recommended that corrective middle ear surgery be evaluated on the basis of percentage of closure of the bone-air gap. We have analyzed all of our results in this way but find the data difficult to interpret. The reason for this is that for tympanoplasty operations: *1.* the ideal result (except for Type I cases) is less than complete closure of the bone-air gap, and

*We are indebted to Dr. Bruce Graham, Dr. Mary Rose Costello and Mr. John Tschantz for performing the audiometric tests.

2. a successful result need not give a hearing gain and may even result in a slight hearing loss. For each type of tympanoplasty, we have determined the number of cases which acquired an absolute level of 30 db, 20 db and 5 db (absolute meaning actual audiometer readings) and the number acquiring a bone-air gap of 30 db or less. All statistical analyses are based on the American Standard Audiometric Zero (see Table III).

TABLE III.—RESULTS.

Type	No. Cases	Acquired 30 db Level (absolute)		Acquired 20 db Level (absolute)		Acquired 5 db Level (absolute)	Bone-air Gap 30 db or Less		Made Worse by 5 db or More	
I	40	33	(82%)	25 (62%) 50% to 15 db		5 (12%)	34	(85%)	4	
II-A	15	10	(67%)	7	(47%)	2	12	(80%)	3	
II-B	2	2		1		0	2		0	
III-A; IV-A	82	39	(48%)	13	(16%)	0	50	(61%)	8	
III-B; IV-B	20	14	(70%)	9	(45%)	0	16	(80%)	1	
V	1	1		1		0	1		0	
	160	99	(62%)	56	(35%)	7	115	(72%)	16	(10%)

TABLE IV.

Case Classification, Based on Bone Conduction (Shambaugh).
Average for 500-1,000-2,000 cps.

Class	Definition	Tympanoplasty Types I	II	III	IV	V	Totals
A	15 db or less	39	14	41	56	1	= 151
B	16 to 25 db	1	1	1	0	0	= 3
C	26 to 35 db	1	1	2	1	0	= 5
D	36 db or more	0	0	0	1	0	= 1
Total		41	16	44	58	1	160

If we utilize the Shambaugh definition of case classifications, we find that nine (6 per cent) of 160 patients had bone conduction averages for 500, 1,000 and 2,000 cps of 15 db or more (see Table IV).

As expected, the best results were for the 40 Type I cases, where 82 per cent acquired a 30 db level and the poorest for the 82 "total plastic" cases (III-A and IV-A), where only 48

per cent acquired a 30 db level. Of the 20 "minor plastic" cases, 70 per cent acquired a 30 db level indicating an improved prognosis when at least half of the ear drum remains. A comparison of our results with those of Zollner is shown in Table V.

A comparison of our results with European authors is valid, of course, only if our audiometers are calibrated to the same Audiometric Zero as theirs. We do not know at this time to what standard the German audiometers are calibrated.

Of the total group of 160 cases, there were 16 (10 per cent)

TABLE V.

Type	Criteria	Zollner (278)	Schuknecht, et al. (160)
I	Bone-air gap 30 db or less	94% (of 52)	85% (of 40)
	Acquired 15 db level or better	62%	50%
	Acquired 5 db level or better	19%	12%
II-A	Bone-air gap 30 db or less	56% (of 39)	80% (of 15)
	Acquired 20 db level or better	27%	47%
III-A IV-A	Bone-air gap 30 db or less	56% (of 133)	61% (of 82)
	Acquired 20 db level or better	23%	16%
III-B IV-B	Bone-air gap of 30 db or less	72%	80%
	Acquired 20 db level or better	42%	45%

which were made worse by 5 db or more. Several of these were worse by less than 15 db and maintained a threshold level better than 30 db.

It should be pointed out that it is sometimes difficult to determine in what type classification a particular case should be placed. For example, if the stapes is present and one-half the tympanic membrane, more or less is usable for reconstruction, should the case be classified as Type III-A or Type III-B? If one author places that case in Type III-A and another in Type III-B, the comparison of these authors' results has little significance. Table VI contains a comparison of our results with those of Wullstein[8] and Proctor.[9]

POSTOPERATIVE FINDINGS.

One of the causes for failure is excessive scarring of the graft to the medial tympanic wall. When this results in an inadequate tympanic space so that the round window is not in an air containing pocket, there is no hearing gain and there may be a hearing loss, depending upon the preoperative levels (see Fig. 19). This unfortunate result is usually caused by epithelial denudation of the hypotympanum whether it be unavoidable, inadvertent or accidental; therefore, it is of great importance to avoid the excessive removal of mucosal polyps and granulations from the hypotympanum, medial tympanic wall and round window niche. It may be preferable

TABLE VI.

Results.

Type	Acquired 30 db level. Wullstein (250)	Proctor (66)	Schuknecht, et al. (119)
II	73% (of 74)	70% (of 23)	80% (of 17)
III	80% (of 133)	58% (of 26 cases)	66% (of 44 cases)
IV	75% (of 43)	69% (of 17 cases)	42% (of 58 cases)

in some cases to allow the inflammatory reaction of the mucosa to subside and introduce the graft at a second operation. It is the opinion of some otologists, however, that multiple operations create excessive scarring. The question as to how to handle these difficult cases has not yet been answered. In our series, there were 20 tympanoplasty failures because of "scarred-down" grafts.

Another cause of failure is necrosis of the graft in the area of the tympanic cavity. This occurred in 14 ears and predominantly in our early cases (see Fig. 20).

Small (occult) perforations existed in 15 ears. These perforations were not visible to the naked eye; however, when controlled air pressure was delivered during the Valsalva maneuver, air was heard to squeak through a small perforation or the graft was seen to flutter at a small point. They

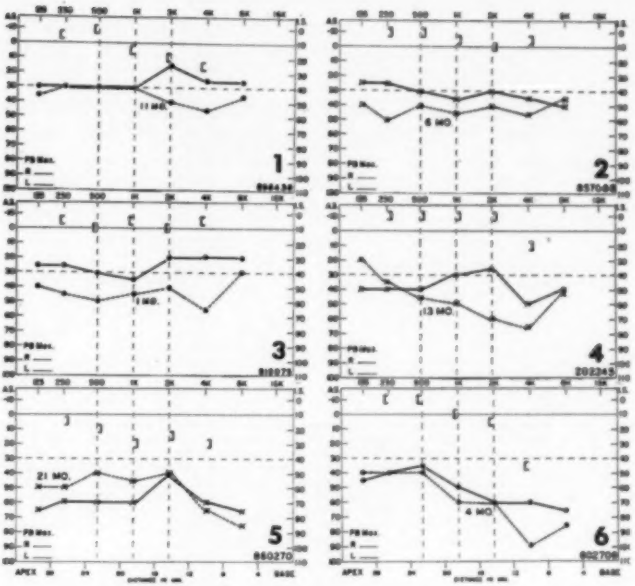

Fig. 19. Failures due to scarred-down grafts. (1) Graft adherent to round window niche, Type III-A. (2) Graft adherent to round window niche because of removal of granulations in that area, Type IV-A. (3) Adhesions of graft to hypotympanum, Type IV-A. (4) Graft adherent to round window niche, Type IV-A. (5) Extensive scarring of graft to hypotympanum, Type IV-A. All of these ears suffered advanced soft tissue changes in the mesotympanic and hypotympanic areas.

do not appear to affect the hearing result. Only time will tell whether these small perforations will result in deterioration of the present satisfactory postoperative state. There was recurrence of middle ear cholesteatoma in three patients, all of whom have been recently reoperated.

There was postoperative fluctuation of hearing in three patients. This appeared to result from relaxed grafts which were made to bulge away from the round window niche on inflation but after a few seconds or minutes returned to their original position thereby eliminating again the sound pressure differential. To avoid this complication, the operative technique should be directed at providing adequate depth to the

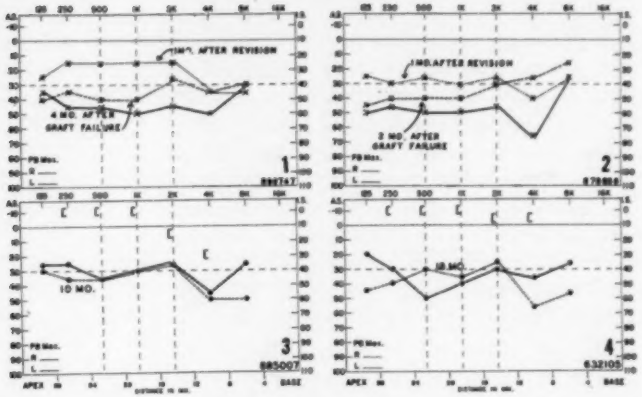

Fig. 20. Examples of failure because of partial graft necrosis. (1) Type III-A with mastoidectomy, subsequent successful reoperation. (2) Type III-A with mastoidectomy, subsequent successful reoperation. (3) Type I, recently reoperated successfully. (4) Type IV-A with mastoidectomy, to be reoperated. Cause for graft necrosis appear to be multiple: (1) Improper fitting and approximation of graft to its bed. (2) Inadequate packing. (3) Failure to remove underlying disease. (4) Failure to prepare a properly de-epithelialized bed. (5) Infection. At reoperation, the old graft must be completely removed and replaced.

TABLE VII.

Postoperative Findings (160 Cases).	
Scarred down graft	20
Partial necrosis of graft	14
Minute (occult) perforations	15
Recurrence of cholesteatoma	3
Fluctuating hearing	3
Continued suppuration	4

cavum minor. Thus, the annulus should be preserved inferiorly and the graft stretched across the tympanic space.

Of 136 patients with a follow-up period of three months or longer, four continued to have discharging mastoid cavities (see Table VII).

Bone conduction losses were experienced in four ears (see Fig. 21). In one, the loss may have been due to a small perforation in the footplate occurring when tympanosclerotic material was removed from the oval window. In the others,

the reason for the bone conduction losses remain unexplained. In two, the losses were progressive over a period of months following surgery. They were not associated with vestibular symptoms and, therefore, are probably not due to diffuse serous labyrinthitis. One might speculate that the operation in some way has promoted the absorption of toxic material through the round window membrane which has damaged the sense organ.

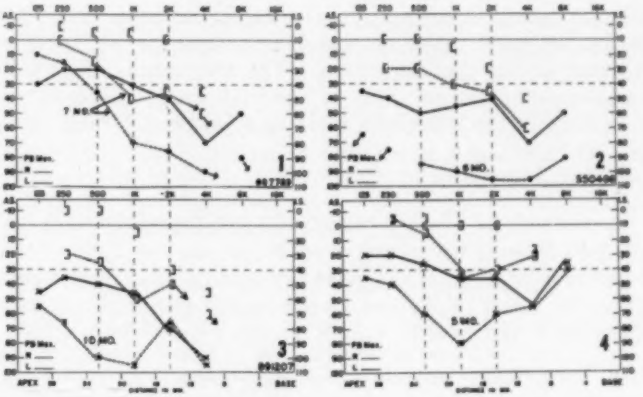

Fig. 21. Sensory-neural losses following tympanoplasty. (1) Type I with successful grafting. The loss for bone conduction thresholds was slowly progressive over a seven-month period. (2) Type III-A with mastoidectomy. In this case, the bone conduction loss plus a scarred down graft has resulted in a severe hearing loss. (3) Type IV-A with mastoidectomy. (4) Type IV-A with mastoidectomy. There was advanced tympanosclerosis of the promontory, sinus tympani, facial ridge and filling of the oval window. The ossicles except for the footplate were missing. A small perforation was made inadvertently in the footplate as otosclerotic material was removed in this region and may be the cause for the bone conduction loss in this case.

CONCLUSIONS.

Tympanoplasty surgery is an exercise in micro-surgical technique which requires good judgment, great patience and an understanding of the physiological principles of sound transmission.

Success depends upon meticulous removal of diseased tissues and reconstruction of the tympanic space so as to accomplish

a columnella effect and/or a pressure differential at the windows.

It is most important that the mucosa of the tympanum, medial tympanic wall and round window area be cautiously preserved and that the skin graft, preferably full-thickness from the post-auricular sulcus, be trimmed, fitted and packed into place with great care.

Ten per cent of the patients were made worse by 5 db or more, but several of these losses were less than 15 db and have been corrected subsequently by surgical revision. The percentage of good cases increases with time as our surgical technical ability has improved. The two main reasons for tympanoplasty failure are: *1.* partial graft necrosis, and *2.* scarring of the graft into the hypotympanum. Four of the patients have had a significant unexplained loss of bone conduction thresholds.

An analysis of the results of 160 tympanoplasties reveals that a 30 db level was acquired by 82 per cent of Type I cases, 70 per cent of Type II cases, 48 per cent of total plastic cases (Type III-A and IV-A) and 70 per cent of minor plastic cases (Type III-B and IV-B).

Our experience has shown that damage to ossicles and tympanic membranes was less in the younger age group and the success of tympanoplasty operations depends in part upon early intervention in chronic irreversible suppurative middle ear disease.

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INCUDOSTAPEDIAL JOINT SEPARATION: ETIOLOGY, TREATMENT AND SIGNIFICANCE.*

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ETIOLOGY OF INCUDOSTAPEDIAL JOINT SEPARATION.

I. Incudostapedial Joint Separation from Congenital Causes.

1. The malleus and incus are derived from the first visceral arch (see Fig. 1). Improper differentiation of this arch may cause distortion or absence of the incus. When other anomalies are not present, a total absence of the incus is considered to be quite rare. I have found only one case of total absence of the incus thought to be of congenital etiology (see Fig. 2).[12]

2. Improper differentiation of only the long process of the incus can produce the functional effect of incudostapedial joint separation.[13] Fig. 3 is a photograph presenting this condition as found in a ten-year-old child with conductive deafness, which I considered to be congenital. Tympanotomy revealed a distorted thick stapes with congenital fixation of the footplate. The incus was nothing more than a band of heavy connective tissue without bony content.

3. Embryologically, the incus is derived, as previously stated, from the first visceral arch whereas the stapes is derived from the second visceral arch (see Fig. 4). Contact of the lenticular process of the incus with the head of the stapes and consequent formation of the incudostapedial joint occurs in the eighth week of fetal life. The attraction of these two bony surfaces for each other is phenomenal and unparalleled in the human body. It is amazing that they so uniformly come together and join in an articulation. The congenital abnormality of lack of articulation of these two

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bones should not be rare; but, strangely enough, no true proven case has been reported to my knowledge. One would expect this to be one of the most common congenital anomalies found in the human body.

4. A disturbance in formation of the incudostapedial joint may involve other structures, such as the stapedius muscle and the head of the malleus. An example of this is a patient, whom I have previously presented,[13] with triple bony union of the malleus, incus and stapes and absence of the stapedius muscle, tendon and pyramidal eminence (see Fig. 5).

II. Incudostapedial Joint Separation Due to Surgical Procedures.

Today, this is perhaps the most common cause of interruption of the ossicular chain. Separation of this joint has occurred in innumerable cases as the result of surgical maneuvers during the operation for mobilization of the stapes. Fortunately, repositioning at the time of surgery can many times be accomplished, but often the lenticular process has been irreparably damaged and a true incudostapedial joint separation results (see Figs. 6 and 6-a).

Other surgical procedures about the mastoid and middle ear may also cause dislocation. One authority reported one case[27] and another authority[31] recently reported three cases, in which there was a dislodgement of the incus from its epitympanic and stapedial attachments during a previous simple mastoidectomy.

III. Incudostapedial Joint Separation Due to Imflammatory Necrosis of the Long Process of the Incus.

This is a very commonly observed pathologic cause of incudostapedial joint separation (see Fig. 8). The long process of the incus is more vulnerable to damage from inflammation than any other portion of the ossicular chain.[13] The major reason for this is the fact that the long process of the incus projects for a considerable distance into an air-containing cavity and is a longer distance away from soft tissue blood supply than any other portion of the ossicular chain. Thus, an acute inflammatory process of the middle ear may be con-

trolled after it has caused necrotic destruction of the long process of the incus. The remainder of the middle ear, as well as the tympanic membrane, may return to a normal state. In such a case, the loss of function will be almost entirely due to interruption of the ossicular chain, and the hearing will be at a near 60 db threshold. If a tympanotomy is done, a normal tympanic membrane will be encountered, and when elevated the structures of the middle ear may be curiously free of any evidence of previous disease with the one exception of destruction from necrosis of the long process of the incus.

IV. Incudostapedial Joint Separation Due to Head Injury.

Much exceptionally fine investigative work has been directed toward the effects of injury on the ear.[2,3,17] Detailed analyses of the effects of concussion and skull fracture have been brilliantly presented by many contributors.[10,11,12,18,19,20,24,25,39] From an audiologic standpoint, the chief finding in these cases reported was that of perceptive loss from cochlear damage. We now know that the conductive loss of hearing in head injury is quite common[13] (see Audiogram I).

On February 7, 1957, I did a tympanotomy on a patient with the anticipation of doing a mobilization of the stapes.[13] This patient had a 67 db pure tone loss in the speech frequencies with a near normal bone conduction. On entering the middle ear, the stapes was found to be quite mobile, but there was a total incudostapedial joint separation (see Fig. 9). This patient had given a history of having suffered a severe blow on his head during his youth and had stated that he believed the onset of his hearing loss to be at that time. Head injury, producing marked cochlear damage, could easily be explained by information reported by previous investigators, but ossicular disarticulation had been very rare even in crushing head injuries producing death. I reported this case, but, although the history was strongly suggestive, I held at that time some reservation in stating that trauma was definitely the etiologic factor. Since that time I have encountered two more cases of unilateral conductive deafness due to incudostapedial joint separation produced by skull fracture. In these cases, there was little doubt as to the history.

One authority recently reported one case of incudostapedial joint separation apparently due to a non-fracturing blow on the head.[21] Another authority recently reported a case of bilateral incudostapedial joint separation due to severe head injury.[9] Several other cases of conductive deafness, probably due to ossicular chain separation from trauma to the head, have been reported to me by personal communication.[5,29] A few of these have been proven; others are awaiting exploratory tympanotomy. I now have two cases awaiting operation which might well fall into this group.

I am sure that this cause of deafness is not as rare as is suggested by the lack of evidence in the literature. On the contrary, the fact that I have seen this many cases suggests that injury is not an uncommon cause of incudostapedial joint separation.

Several factors will make this condition more frequently observed in the future.

1. More tympanic cavities will be explored when unexplained conductive deafness is found in either one or both ears.

2. In this age more head injuries will occur from high speed vehicles and industrial accidents.

3. More severely injured patients will be kept alive by modern surgical and medical measures.

4. By realizing that a conductive deafness may result from this cause, otologists will doubtless find and correct this disability in more patients in the future.

DISCUSSION OF TRAUMA TO THE HEAD IN RELATION TO INCUDOSTAPEDIAL JOINT SEPARATION.

Immediately, our thinking is arrested by the query as to what might possibly have happened to cause separation of this joint during these traumatic moments when the injury to the head is occurring.

1. An important factor to be considered in incudostapedial joint separation due to injury of the head is the effect of

concussion. A severe vibratory reaction to an impact sufficient to produce fracture of the skull may cause momentary separation and weakening of the tissues with loss of elasticity. The cohesiveness of the tissues is temporarily disrupted as though there had been an explosion among the cells.

2. Still another factor is the law of physics related to inertia. Newton's law of motion related to inertia states that "A body at rest will remain at rest and a body in motion will continue in motion with constant speed in a straight line, as long as no unbalanced force acts upon it."[28]

When the head is in motion and strikes a stationary object, all of the head tends to continue in motion (see Fig. 23). The moveable objects within the head, *e.g.*, brain, ossicles, etc., must react in a physical way to acceleration and deceleration. Even when the head is stopped, the brain, ossicles, etc., must be under a strong force to remain in motion in accordance with the laws of inertia. In an opposite way, the same is true when the head is struck by a moving object. This factor in incudostapedial joint separation is mentioned here for completeness of thought, but consideration of these facts is not considered desirable in this publication.

3. The action of the tympanic muscles must be considered.

The two tympanic muscles are partially counter-active in the force they exert on the ossicular chain. This permits delicate regulation of its rigidity in suspension.

The structural arrangement of the muscular fibers are of a pennate type which allow some fibers within the muscle to either release or neutralize others. This partially explains why the tensions produced by these muscles are less proportionately than would be expected from their size. Nevertheless, the linear force exerted by the stapedius muscle is considerable.

Investigators have attempted to determine the maximum tension of which these muscles are capable. One investigator estimated that the tensor tympani in a rabbit exerted approximately 1.2 gm. pull when stimulated by sound.[22] Other investigators estimated that the stapedius muscle of a cat exerted

about 1 gm. tension under experimental conditions.[39,40] This would represent a substantial force in the human. Another investigator found that the human stapes could be pulled back for a distance of 200 micra by a simple acoustic reflex.[40] The anterior portion of the annular ligament was pulled back and stretched 60 micra. The inward motion of the footplate of the stapes was measured in a rabbit by one authority to be 0.027 mm. on acoustic stimulation.[40]

Although the stapedius muscle is extremely small, if it were thrown into sudden severe tetanic contraction by severe injury (trauma to the head), the total force would be exerted against the smallest joint in the human body. Even large bones are occasionally fractured in this way during shock therapy.

The pull of the tensor tympani producing a firm medial thrust of the incus with a simultaneous exceptional posterior pull on the head of the stapes from the stapedius muscle action might be a factor in producing disarticulation of the joint.

Another factor may be the twisting of the incudostapedial joint. Some authorities consider the ear to have two axes of rotation.[23] One axis is along a line through the short process of the incus and the anterior ligaments of the malleus; the other, from the head of the stapes to the footplate.

Incudomallealar articulation divides the two large ossicles into two portions of equal weight.[23] Along this axis is a line where the moment of inertia of the ossicles is at its lowest value. The weights above and below are equal. The upper mass acts as a counter-weight on the lower mass.

Utilizing certain laws of physics, it can be demonstrated that the incudostapedial joint acts much like the well-known "universal joint" because it lies between the two major axes of rotation. As the "universal joint" in a machine prevents a disastrous play of unbalanced counter-forces from producing destruction or distortion during the transmission of energy, likewise the incudostapedial joint performs this vital role in sound transmission.

This "universal joint" concept is related to the incudostapedial joint separation in the following way: If a low

frequency tone (10 cycle) is administered to the normal ear in increasing intensity, the perceived loudness will increase as expected; however, at a certain critical point, contrary to expectation, further increase of intensity will result not in increase in loudness, but in decrease. This indicates that a protective mechanism has come into action. It has been thought that the entire mode of action of the ossicles changes at this time. The stapes stops rotating about the vertical axis, which produces an ever increasing pressure against the cochlea. Instead, the stapes turns in a rotary action at right angles to the previous axis. This is thought to cause the labyrinthine fluid to flow from one edge of the footplate of the stapes to the other in an ineffective manner, thus protecting the cochlea from trauma.

Thus, we can see the importance of this incudostapedial "universal joint" in allowing the structure of the middle ear to vibrate in two different modes with ability to transfer from one mode to the other when a sudden need arises. This ability may, however, add to the vulnerability of the incudostapedial joint to separation. A sudden severe vibration may cause this twisting shearing motion, and in combination with the other forces of trauma to the head may cause the joint to become separated.

In summarizing, these may be some of the factors producing incudostapedial joint separation in trauma to the head:

1. The presence of tissue weakness due to the explosive effect of concussion.

2. The simultaneous jarring effect, tending to produce derangement of all moveable parts according to the laws of physics related to inertia.

3. A sudden severe tetanic contraction of the tympanic muscles with an abrupt change in the axis of rotation of the ossicles.

TREATMENT OF INCUDOSTAPEDIAL JOINT SEPARATION.

Several methods of restoration of hearing following incudostapedial joint separation need to be considered.

I. STAPEDIOPEXY.

This is the surgical procedure used in most of the cases of incudostapedial joint separation which have been previously reported in the literature.[27,31,33] Stapediopexy must remain as the cornerstone for restoration of the transmission of sound from the tympanic membrane to the cochlea when this joint is separated; nevertheless, there are several disadvantages.

Disadvantages of Stapediopexy.

1. Stapediopexy is frequently a major operative procedure and results in considerable morbidity.

2. Stapediopexy frequently requires an endaural incision with removal of enough of the posterior superior bony canal wall to allow a tympanomeatal flap to be brought down into contact with the head of the stapes. If removal of a considerable amount of bone in this area is needed, a cavity is created which may lose its ability to cleanse itself and thus require continuous observation.

3. In stapediopexy, it may be necessary to cut the chorda tympani nerve in order to bring the tympanomeatal flap medially against the head of the stapes.

4. The handle of the malleus is located near the center of the tympanic membrane, and is designed to collect the energy of sound without loss of efficiency. If the head of the stapes is to receive this energy directly, one must consider the fact that it is not located in the most efficient area of the tympanic membrane; furthermore, unless the handle of the malleus is dissected from the drum and removed, it will act as a buffer and tend to dampen and dissipate sound. This loss is admittedly small, but even 2 to 3 db is worth infinite care in preserving when the end-result may be near the critical 30 db level.

5. In stapediopexy, there is no assurance that the head of the stapes will adhere to the mucous membrane lining of the tympanic membrane. I have found it mechanically quite difficult to denude the inner surface of the tympanic membrane in the desired area. This single objection, however, can easily

be overcome by a simple procedure which I now frequently employ in cases which require stapediopexy.

Procedure to Insure Union of the Stapes Head to the Tympanic Membrane.

1. Mobilize the tympanomeatal flap so that it will come into contact with the head of the stapes (see Fig. 17). This may require a wide exposure and the removal of a considerable amount of bone of the posterior superior canal wall. In some patients, a narrow field exposure may be all that is necessary. A simple shifting of the tympanic membrane in the posterior superior quadrant, medially to the head of the stapes, may be accomplished. The depth of the middle ear will be the determining factor.

2. Locate the point of contact of the tympanic membrane with the head of the stapes. At this point make a small incision through the eardrum with a knife or pick (see Fig. 18). Roughen the head of the stapes with a pick.

3. Insert the head of the stapes into the newly made hole in the tympanic membrane (see Fig. 19) and place a small piece of gelfoam over it (see Fig. 20). Follow this by a routine dressing to the external canal.

This procedure insures a fibrous union of the tympanic membrane with the head of the stapes in exactly the desired location.

The above procedure is used only in cases of an intact eardrum.

Those cases which require skin graft, of course, need not have this operation to insure adherence.

In spite of the above disadvantages, stapediopexy is one of the most successful methods for restoration of hearing in cases of incudostapedial joint separation.[15,16] At the present time, stapediopexy occupies a position of usefulness in those cases where it is impossible to use the incus or malleus in the reconstruction of the conductive bridge.

II. REPOSITIONING OF THE LENTICULAR PROCESS OF THE INCUS ON THE HEAD OF THE STAPES.

The second procedure for the repair of incudostapedial joint separation is the repositioning of the lenticular process of the incus on the head of the stapes. Due to the number of operations for mobilization of the stapes being done, this is undoubtedly the most frequently performed procedure for repair of incudostapedial joint separation and is the simplest. Figs. 12 and 13 are photographs of a patient with a fracture of the skull. Please note the skull fracture in the posterior superior canal wall. Repositioning is the procedure of choice in those cases of mechanical separation of the incudostapedial joint in which natural apposition is easily attained. Fortunately, healing of this joint by union with soft tissue is usually dependable.[2,4] Repositioning was possible here with a good result (see Fig. 14, Audiogram III). The type of joint thus established in the process of repair is unknown,[2] but study of this with experimental animals is now beginning.

III. THE USE OF AUTOGENOUS BONE GRAFT FOR THE REPAIR OF INCUDOSTAPEDIAL JOINT SEPARATION.

The third procedure which may be used to repair an incudostapedial joint separation is the use of an autogenous graft of bone. The use of a small bone graft between the widely separated joint surfaces of the incus and stapes was previously reported by the author[13] (see Figs. 9, 10, 11; Audiogram II). This method has been employed on numerous occasions before and since, and has been uniformly and apparently permanently successful. I recommend it as an amazingly simple and effective procedure in the treatment of this condition.

Technique.

1. Make a routine mobilization of the stapes incision and expose the middle ear in the incudostapedial joint area. It is easy to continue the incision if a wide endaural approach for stapediopexy is found to be needed (see Fig. 17).

2. Roughen with a pick the joint surface of the lenticular process of the incus and the articulating surface of the head of the stapes (see Fig. 9).

3. Remove a curette full of bone from the posterior superior canal wall to be used as a bone graft (see Fig. 21). An attempt is made to take a piece of bone approximately twice as large as the area needed to be grafted.

4. With picks, tease the small bone graft between the articulating surfaces of the incus and stapes to fill the area of separation (see Figs. 7, 11, 16). Hearing is usually restored instantaneously (see Audiograms II, IV, V).

Fig. 9 is a photograph of a patient with a head injury. Audiogram I demonstrates the hearing level prior to joint restoration. Fig. 11 is a photograph of the bone graft in place. Audiogram II represents the restoration of hearing in this patient two years after reconstruction. Fig. 15 is that of a photograph of joint separation from fracture of the skull. Fig. 15-a is a supermagnification of this separation. The photograph in Fig. 16 demonstrates repair by graft of bone from the posterior superior canal wall. Results of this repair are shown on the audiogram one year later (Audiogram IV). Fig. 6 is a photograph of a patient one year after stapes mobilization. It is noted that there has been necrosis of the lenticular process of the incus with separation of the incus from the stapes. Fig. 6-a is a super-magnification of this separation. The photograph in Fig. 7 demonstrates a repair by graft of bone from the posterior superior canal wall. Hearing was restored to a good serviceable level following this reconstructive surgery (see Audiogram V).

DISCUSSION OF THE USE OF BONE GRAFTS IN THE MIDDLE EAR.

If bone grafts will produce a functional result when inserted into an air-containing cavity and situated at a point relatively remote from an abundant blood supply, it is the author's opinion that autogenous bone grafts can be used in many ways to repair conductive lesions of the middle ear.

One may ask, what are the end-results of healing with bone grafts of this type? It is unknown whether or not this bone remains viable as bone. It may change into thick fibrous tissue. In either case, it is apparent that it is efficient in restoring this joint to a peak of maximum conductive ef-

INCUDOSTAPEDIAL JOINT SEPARATION

J. V. D. Hough

PRESENTATION OF PHOTOGRAPHS AND DRAWINGS

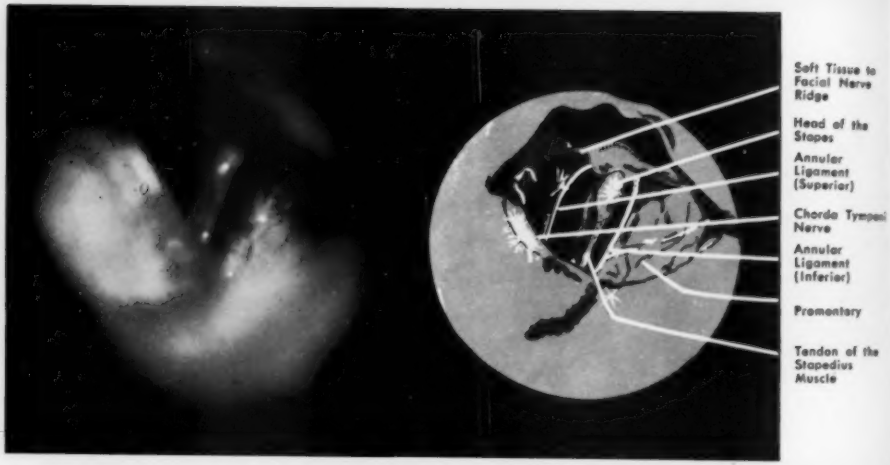


Figure 2.

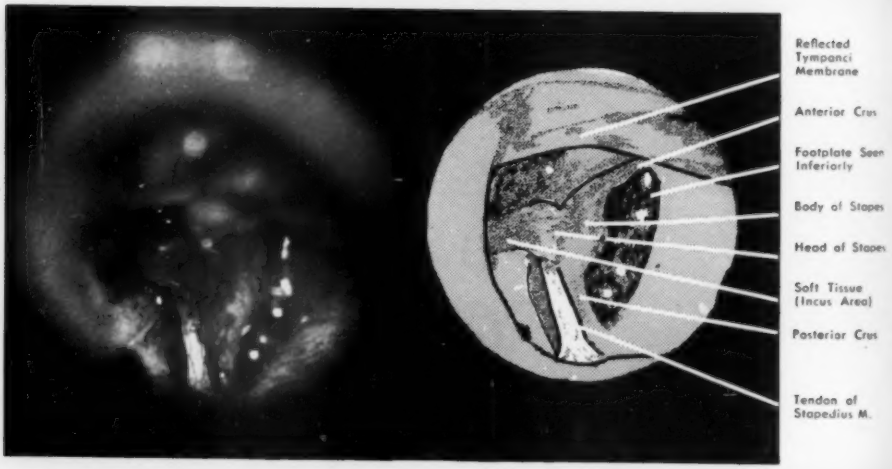
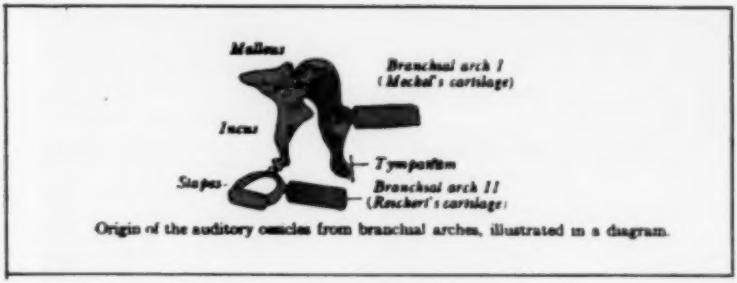


Figure 3.



Origin of the auditory ossicles from branchial arches, illustrated in a diagram.

Figure 1

Figure 1

Drawing from diagram in Arey, "Developmental Anatomy," W. B. Saunders Co., 1940.

Figure 2

Photograph of right middle ear displayed for the purpose of showing a total absence of the incus thought to be of congenital origin.

Due to a profound conductive deafness in this ear, this patient was thought to have had otosclerosis. No other congenital anomalies could be found and there was no history or other evidence of middle ear pathology.

Tympanotomy revealed a total absence of the incus. The soft tissue strand extending superiorly to the facial ridge and inferiorly to the promontory is thought to be connective tissue from residual mesenchymal tissue. This, perhaps, is the tissue which should have differentiated into the long process of the incus. It is without bony content. The stapes was not fixed and the round window and handle of the malleus appeared normal.

Lack of proper specific differentiation of the incus portion of the first branchial arch apparently produced this anomaly. This is significant in that it produces the functional impairment of ossicular chain separation. Restoration of function was accomplished by stapediopexy.

Figure 3

Photograph of the right middle ear displayed for the purpose of showing a congenital abnormality which can produce the functional result of ossicular chain separation. This is the ear of a 10 year old child who has had a conductive deafness presumably since birth.

Tympanotomy here revealed a soft tissue mass coming from the epitympanic region which attached to the stapes head. This tissue was without bony content and gave the functional result of an absence of the incus. Please note the deformed stapes. The body is much thicker than usual and the crura are flattened toward the footplate. The inferior portion of the footplate is easily seen but a definite annular ligament is not identifiable. This stapes was congenitally fixed and represents a condition of improper differentiation of the lamina stapedialis.

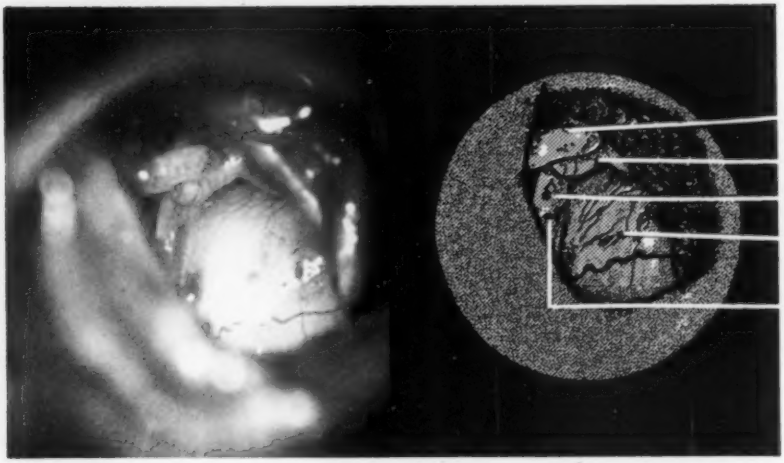


Figure 5.

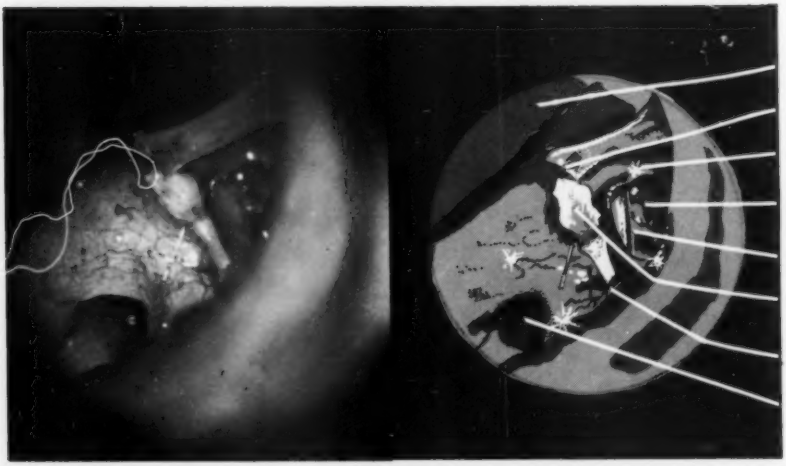


Figure 6.

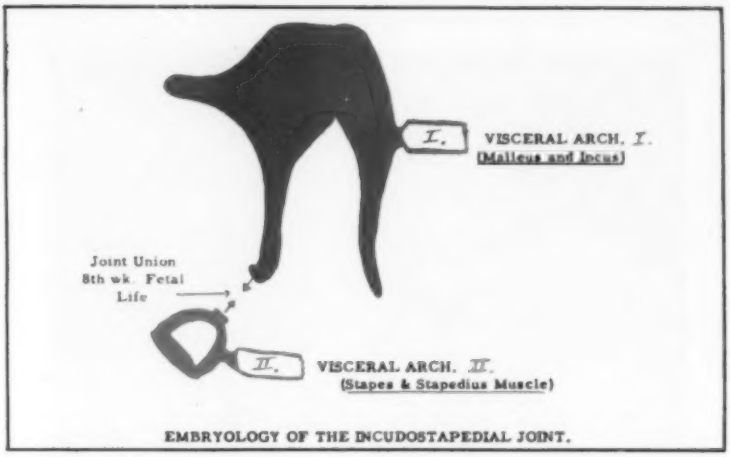


Figure 4.

Figure 5

Photograph of the right middle ear presented for the purpose of demonstrating ossicular bony malformations which can produce a functional disturbance in sound transmission along the ossicular chain. This ear has the rare congenital malformation of triple union of the malleus, incus and stapes.

There is a total absence of the stapedius muscle, tendon and pyramidal eminence in this ear. The posterior crus of the stapes is seen but the view of the anterior crus is obstructed by the triple bony union. The twisted S-shaped long process of the incus is caught in a bony mass which also includes the head of the stapes and the handle of the malleus. This is clearly seen under and attached to the reflected tympanic membrane.

The problem of micro-orthopedics is obvious. Ossicular separation was accomplished and serviceable hearing was restored.[13]

Figure 6

Photograph of the left middle ear displayed for the purpose of demonstrating an incudostapedial joint separation as a result of previous surgery.

Eleven months prior to this photograph an operation for mobilization of the stapes was performed in this ear by the author. The immediate hearing restoration was excellent but acuity diminished gradually over a period of four months. (Please see Audiogram V).

Almost total incudostapedial joint separation is seen with this tympanotomy. Necrosis of the lenticular process of the incus probably resulted from surgical trauma producing partial interruption of blood supply in this area.

Restoration of hearing was accomplished in this ear by a graft of bone. (Please see Audiogram V and Figure 7).

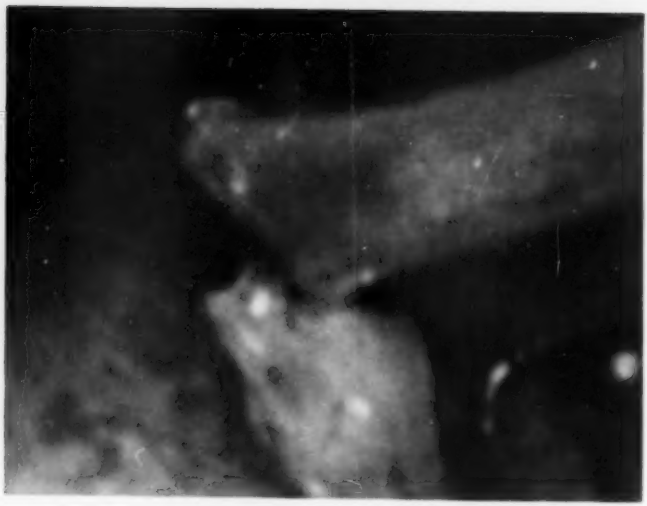


Figure 6A

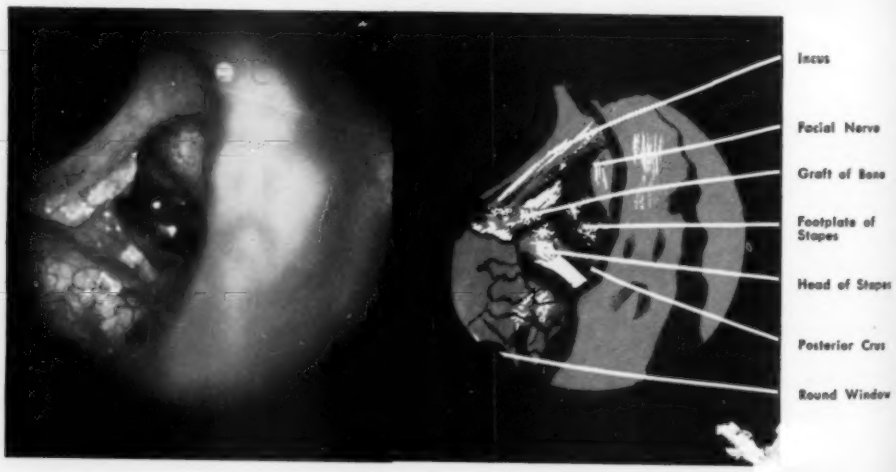
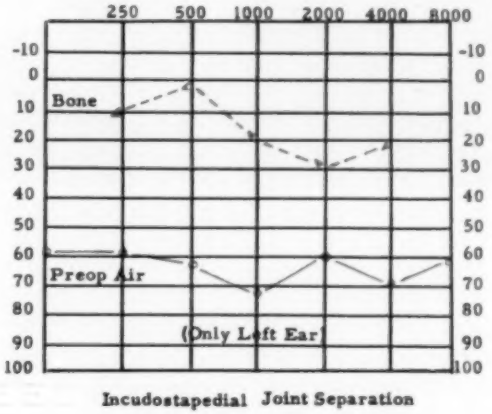


Figure 7.

Figure 6A

This photograph is a supermagnification of the incudostapedial joint separation seen in Figure 6. The tissue repair after trauma resulting in separation of the joint can be seen in greater detail.



Audiogram I

Figure 7

This is a photograph of the same ear as presented in Figure 6 and 6A but showing a repair of the incudostapedial joint separation by a graft of autogenous bone.

After preparing the joint area, a graft of bone was taken from the posterior superior bony canal wall and placed in position between the long process of the incus and the head of the stapes. Immediately hearing was restored to a practical closing of the air bone gap and it has been maintained. (Please see Audiogram V).

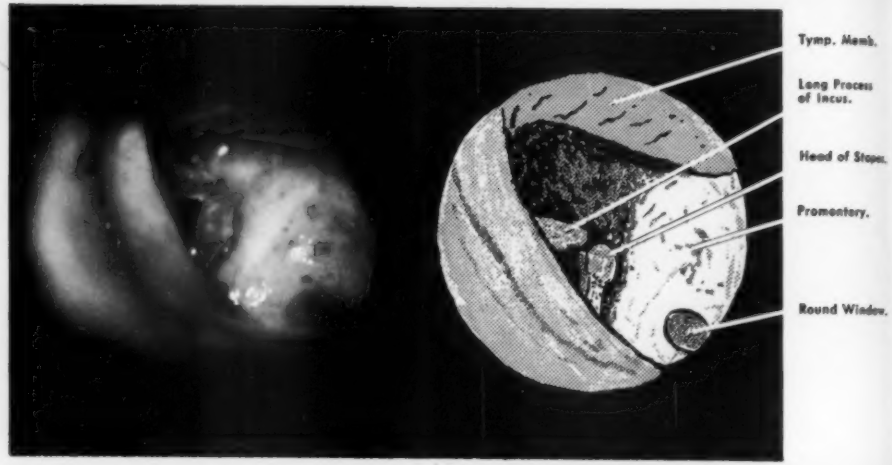


Figure 8.

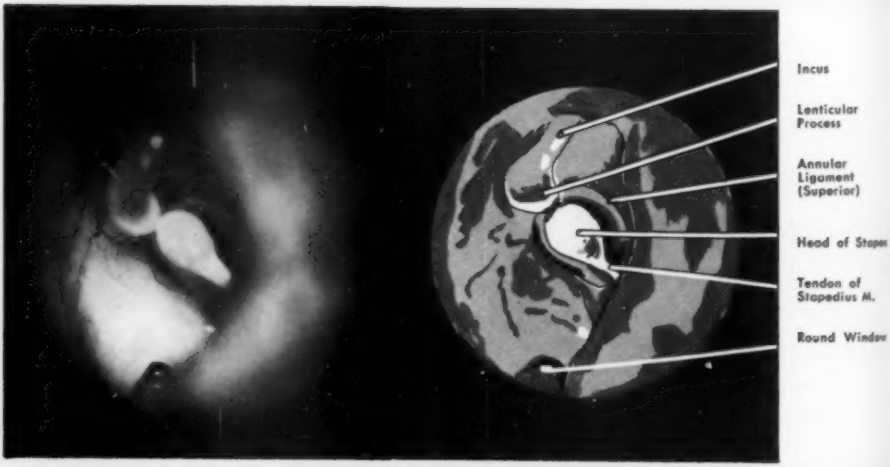


Figure 9.

Figure 8

Photograph of right middle ear displayed for the purpose of showing necrosis of the long process of the incus.

Frequently, the long process of the incus is the recipient of inflammatory destruction. This ear shows a total loss of the lenticular process of the incus. Articulation with the head of the stapes is destroyed. There remains only a small strand of soft tissue between the incus and the stapes.

This type of necrosis is usually believed to be due to inflammation, but frequently this, or a variation of it, will be seen in an ear without history of suppuration. The tympanic membrane may be normal in appearance as it was in this ear. The ossicles may be normal in every other respect. There may be no evidence of adhesion in the middle ear and there may be no other evidence of previous middle ear disease. An early resolution of middle ear disease may have allowed necrosis of this vulnerable long process of the incus and left the remainder of the middle ear undamaged. The hearing loss produced by this is the same as that seen in any other incudostapedial joint separation.

Figure 9

Photograph of the left tympanic cavity displayed for the purpose of showing ossicular disarticulation resulting in complete separation of the incus from the stapes.

This patient suffered a severe blow on his head during his youth and reported that a loss of hearing in this ear began at that time. No other symptoms are present and the opposite ear is functionally normal. Fracture of the skull is thought to be the etiologic factor. The possibilities of seeing this picture as the result of an embryologic disturbance has been discussed in this paper, but the history in this case was quite definite and suggests a traumatic origin.

The functional result of hearing loss in this ear is considered to be the typical finding in incudostapedial joint separation. (Please see Audiogram II).

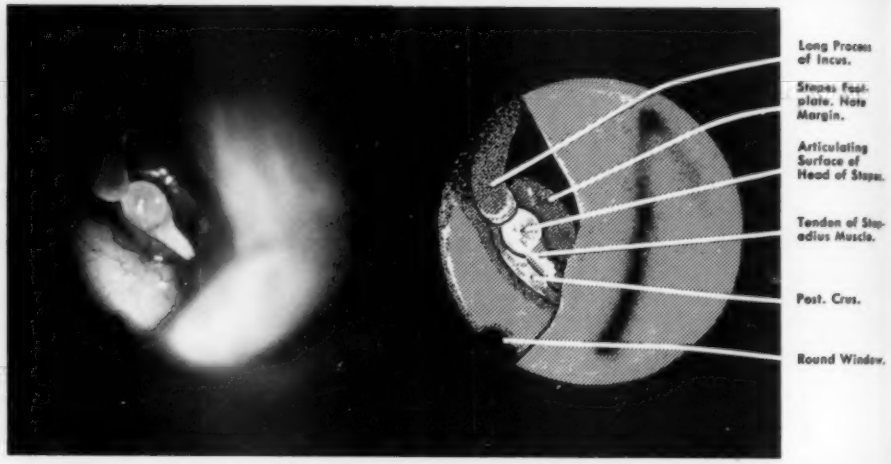


Figure 10.

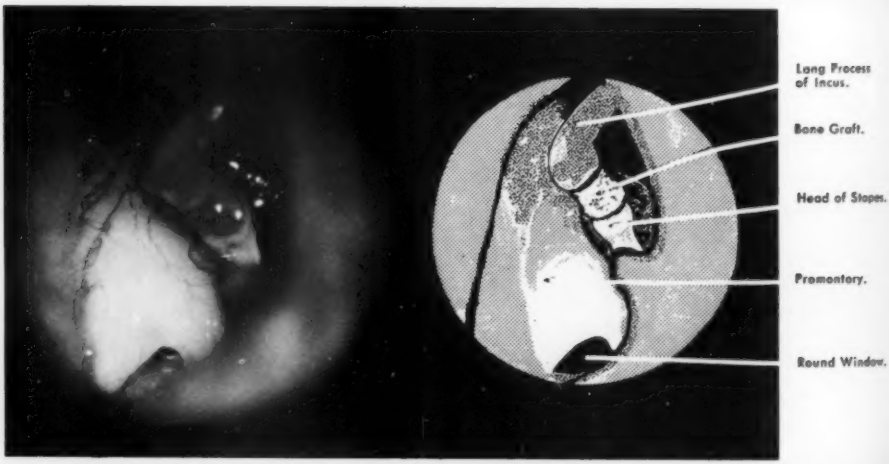


Figure 11.

Figure 10

Photograph of left middle ear (the same as Figure 9). This presents a different view of the stapes and the incudostapedial joint separation with better detail in certain aspects.

This patient had a unilateral purely conductive hearing loss of 67 decibels for pure tone and 62 decibels for speech. No other abnormalities of the middle ear could be found. The round window was present and the membrane visible. The malleus and incus were freely movable. The entire stapes was easily seen. The footplate moved normally in the oval window. There is no evidence of abnormalities of the external or internal ear which would account for the profound loss of hearing. One may assume, with this evidence, that all of the hearing loss was due to a disruption of the ossicular chain. Many observers have suggested that sound reaches the cochlea by molecular transmission through the round window in the theory of "hearing in reverse." Other investigators have suggested that the cochlea receives sound by a universal transmission of energy by all of the surrounding tissues and air without any point of emphasis. These theories deny the pre-eminence of transmission via the tympanic membrane and ossicular chain through the oval window to the cochlea. Some observers ascribe only protective function to the tympanic membrane and the ossicular chain.

This cochlea had the opportunity to receive sound transmitted through either the round window or the oval window. There was no mechanical barrier at either window. Both avenues of entry were perfectly normal; yet there was a loss of 67 decibels for pure tone. This type of hearing loss has been produced experimentally in animals by disruption of the ossicular chain continuity and testing of cochlear potentials by the Wever-Bray phenomenon. This patient, however, represents a human example of the sound transmitting ability of the ossicular chain. Figure 11 will demonstrate the return of that function in the same patient by restoration of the chain.[13]

Figure 11

Photograph of the same left tympanic cavity as seen in Figures 9 and 10, displayed for the purpose of demonstrating operative correction of the incudostapedial joint separation previously seen.

The articulating surfaces of the head of the stapes and the lenticular process of the incus were roughened by the use of small picks. A small shaving of bone was removed from the edge of the bony external canal wall and teased into position between the articulating surfaces of the two ossicles. The hearing was instantly restored to normal and there has been no regression. (Please see Audiogram II).

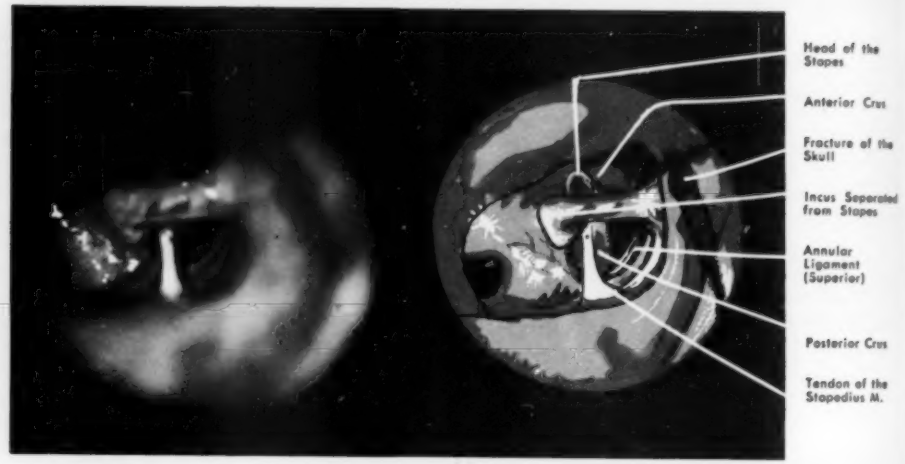


Figure 12.

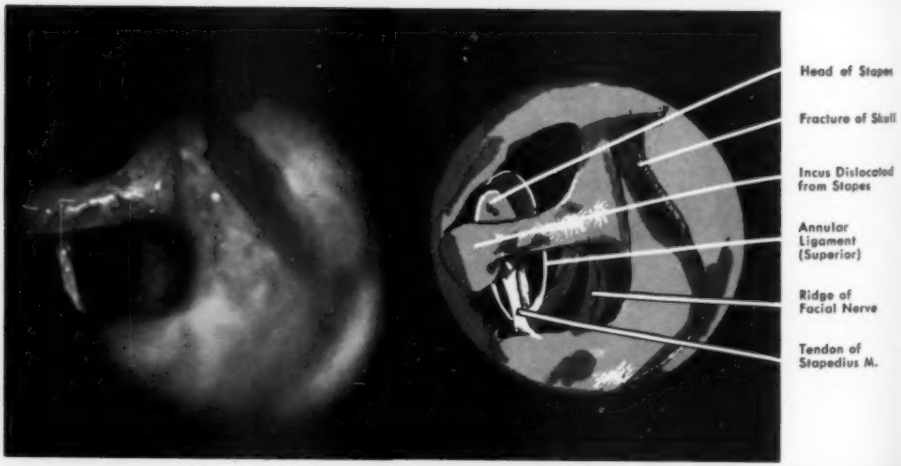
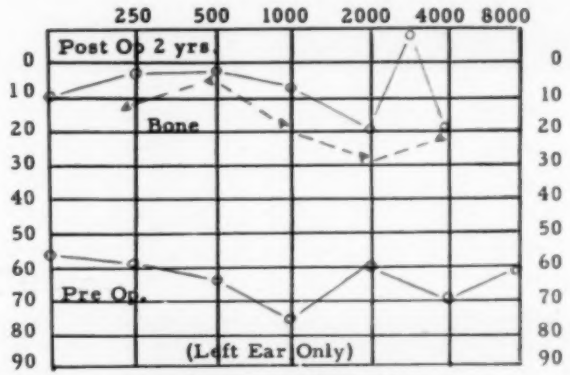


Figure 13.

Figure 12

This is a photograph of the left middle ear presented to demonstrate a recent skull fracture which caused an incudostapedial joint separation. Tympanotomy revealed a large skull fracture extending through the posterior superior bony canal wall. The movement of the fragments may have produced torsion of the body of the incus at the time of injury. Along with other factors mentioned in this paper this may account for the posterior dislocation observed. An excellent view of the entire stapes is seen.

The hearing loss (Please see Audiogram III) in this ear is not the pure picture of incudostapedial joint separation seen in Figure 9 (Audiogram II). This is due to the concommitant cochlear loss as well as the slight transmission of sound vibration through scar bands between the incus and stapes. These bands have been removed prior to the obtaining of this photograph. Another view of this ear is seen in Figure 13.



2 years postoperative
Bone graft to joint separation.

Audiogram II

Figure 13

This is a photograph of the left middle ear of a patient with a fracture of the skull which has an associated incudostapedial joint separation. This photograph is of the same ear presented in Figure 12. A better view of the skull fracture can be seen.

This patient survived an industrial accident which caused a crushing injury of the head. There was a mixed deafness in this ear with a predominantly conductive loss. A diagnosis of incudostapedial joint separation was made before surgery based on the author's previous experiences related in this publication. Restoration of the conductive hearing loss was accomplished by a simple repositioning. (Please see next photograph Figure 14).

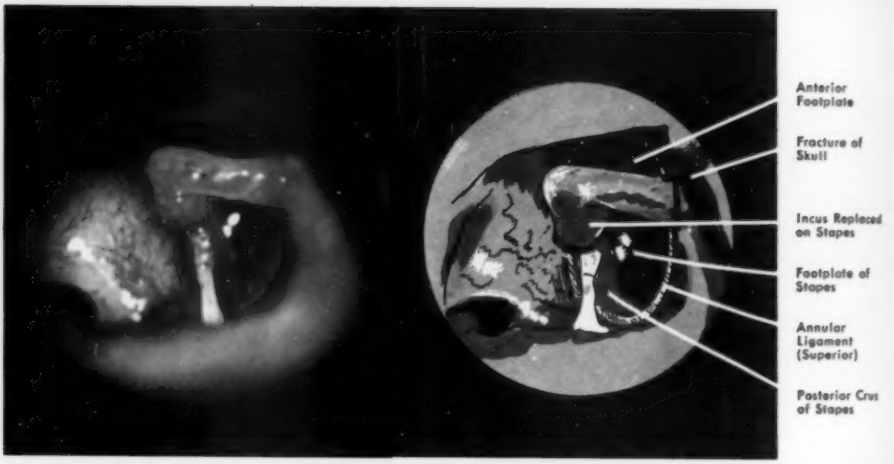


Figure 14.

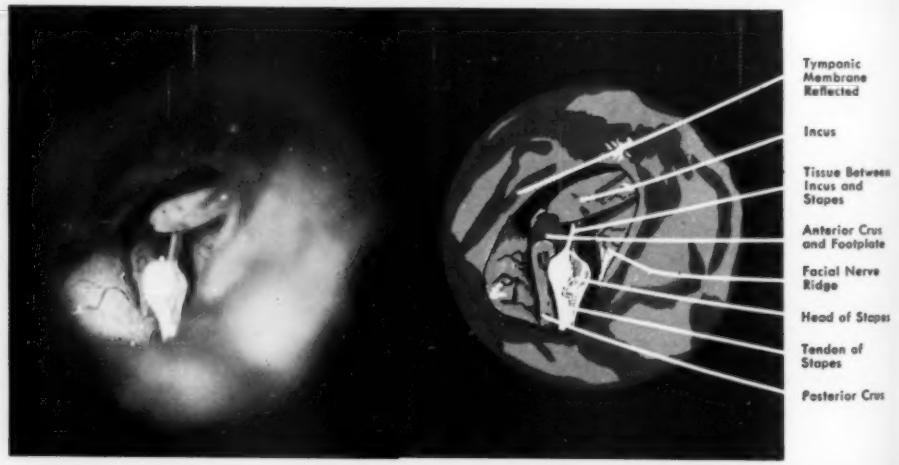


Figure 15.

Figure 14

Photograph of the same ear as seen in Figures 12 and 13 is here presented to demonstrate surgical correction of an incudostapedial joint separation by a simple repositioning.

This surgical procedure was done 10 months after the skull fracture occurred. The immediate gain in hearing resulting from the re-establishment of continuity between the incus and stapes closed the air bone gap. The hearing level has remained stationary since (6 months). (Please see Audiogram III).

Figure 15

Photograph of the left middle ear demonstrating a separation of the incus from the stapes as the result of a skull fracture.

There is a soft tissue strand containing a large blood vessel between the two bones. The anterior crus is entirely visible and the footplate is seen projecting anteriorly a little beyond the crus insertion. This stapes was normally mobile in the oval window.

When this patient was 12 years of age he suffered a severe head injury. At that time he experienced a sudden unilateral loss of hearing in this ear. Preoperative examination revealed a linear scar of the tympanic membrane. At the time of surgery the canal was found to have an old healed fracture obliquely in its posterior superior portion. Tympanotomy revealed an ossicular chain separation of the long process of the incus from the head of the stapes as is seen in the photograph. The lenticular process of the incus seemed to have disappeared during the healing process.

The continuity of the ossicular chain was re-established with the use of an autogenous graft of bone which will be seen in Figure 16.

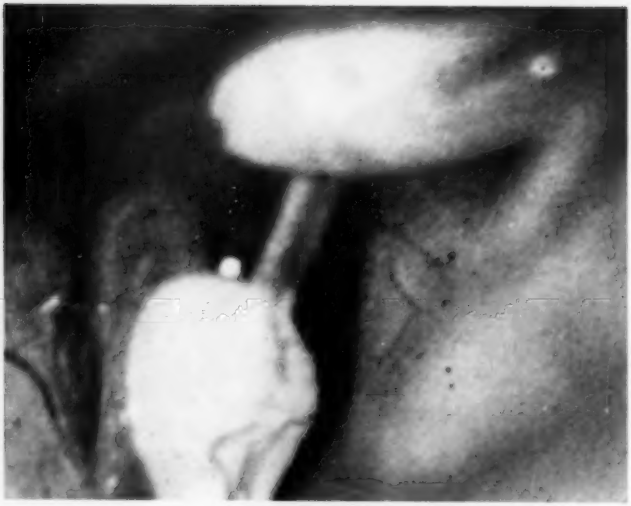


Figure 15A

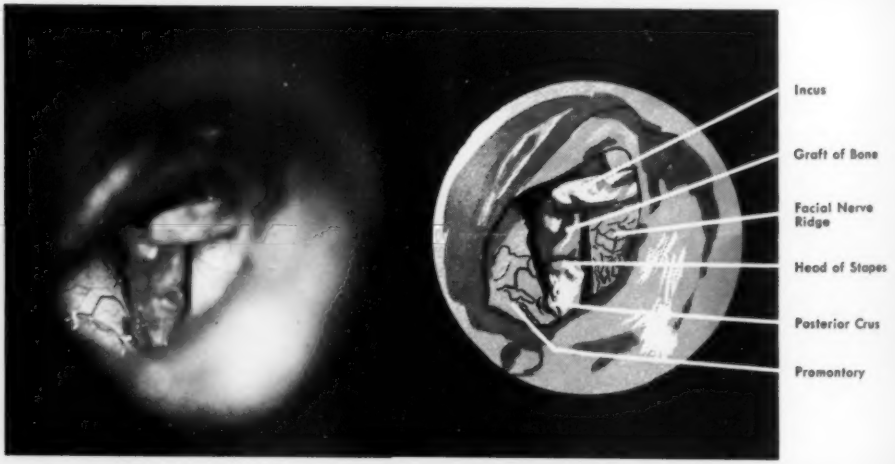
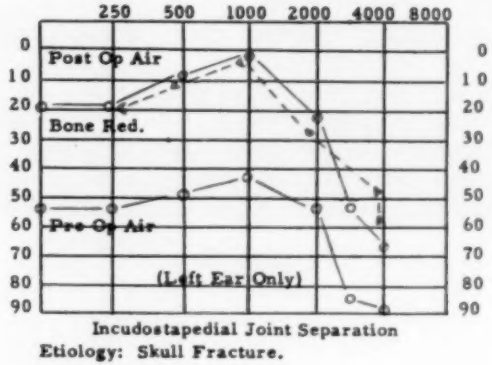


Figure 16.

Figure 15A

Photograph showing a supermagnification of the incudostapedial joint separation seen in Figure 15.



Audiogram III

Figure 16

This photograph is of the same ear as presented in Figure 15 but here demonstrates the surgical restoration of a connecting link between the incus and the stapes.

An autogenous graft of bone was removed from the posterior superior canal edge and placed between the two separated bones. This can be easily identified in the photograph.

Despite the relatively great distance of separation the successful functional result has been maintained for over 1½ years.

The hearing loss following this injury was purely conductive in the lower frequencies but a perceptive loss bilaterally is noted in the higher frequencies. (Please see Audiogram IV). The patient was only 49 years old at the time this operation was done and, therefore, it was believed that the perceptive loss in the higher frequencies was due primarily to cochlear consussion suffered at the time of the original injury and not due to presbyacusis.

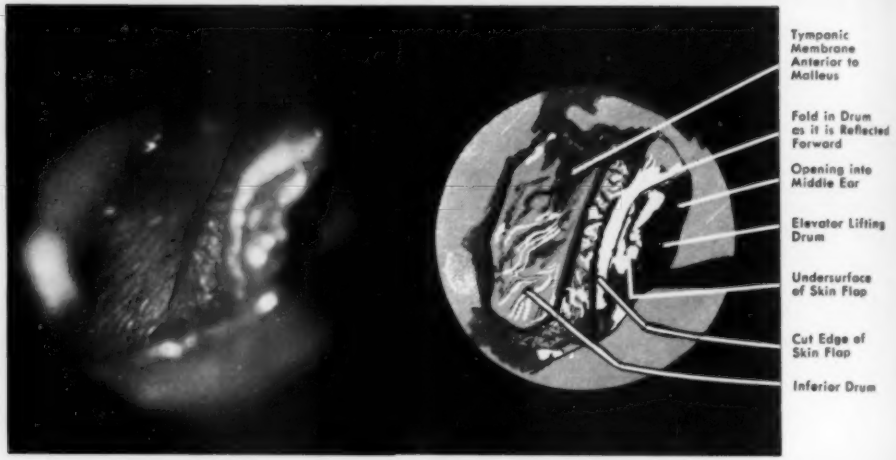


Figure 17.

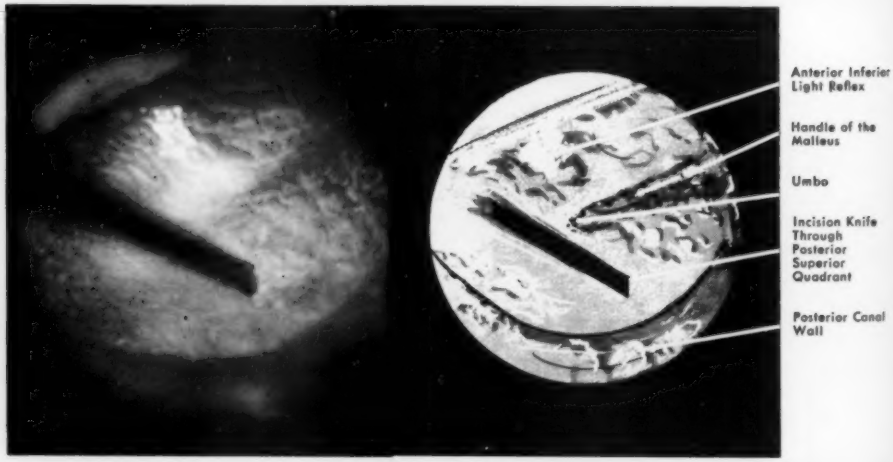
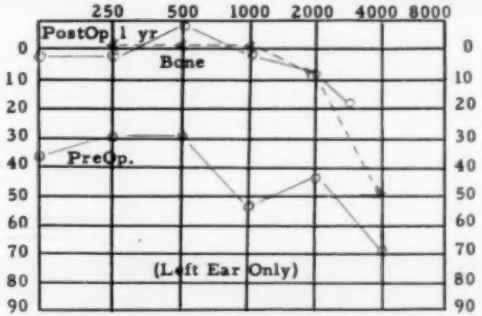


Figure 18.

Figure 17

This photograph demonstrates the typical view seen in the elevation of the skin and tympanic membrane for exposure of the posterior half of the middle ear. The skin and underlying periosteum have been elevated from the incision line in the posterior canal wall down to the tympanic membrane. The middle ear has been entered and the fibrous annulus is being lifted from its sulcus with the elevator.

This exposure used now so frequently for a mobilization of the stapes is an ideal tympanotomy for a repair of incudostapedial joint pathology. If stapediopexy is anticipated, a wider skin flap should be made.



Audiogram IV

Figure 18

This photograph is shown to demonstrate the simple incision in the posterior superior portion of the tympanic membrane. This is done in those cases which require stapediopexy when the drum is intact. The incision is made over the proposed site of union of the stapes head to the tympanic membrane.

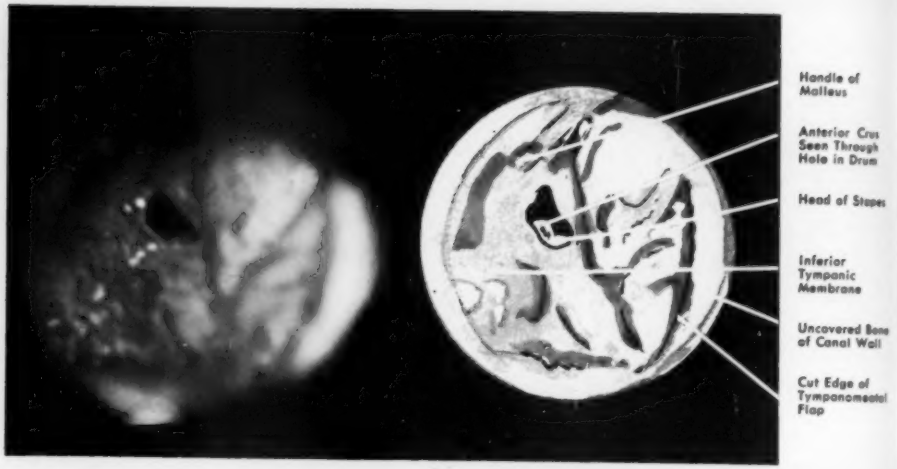


Figure 19.

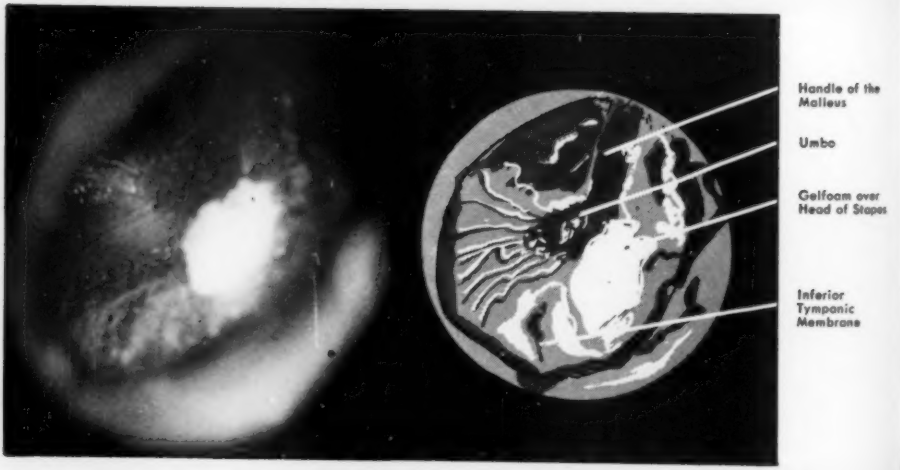
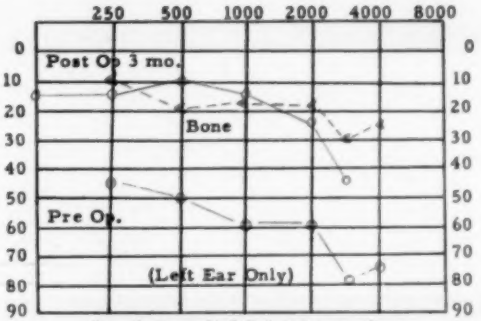


Figure 20.

Figure 19

This photograph demonstrates the placing of the head of the stapes into the incision previously made in the eardrum as represented in Figure 18. Note that the skin flap and upper drum have been allowed to retract in the posterior superior quadrant leaving a small area of uncovered bone on the posterior superior canal wall. The perforation is seen just anterior to its final position wherein it will become a "collar" for the head of the stapes. This will insure permanent union of the tympanic membrane to the head of the stapes.



Incudostapedial Joint Separation
Repair with Bone Graft.

Figure 20

This photograph presents the end of the stapediopexy procedure in which the stapes head has been placed through an incision in the eardrum. Gelfoam has been placed over the area where the head of the stapes projects through the perforation.

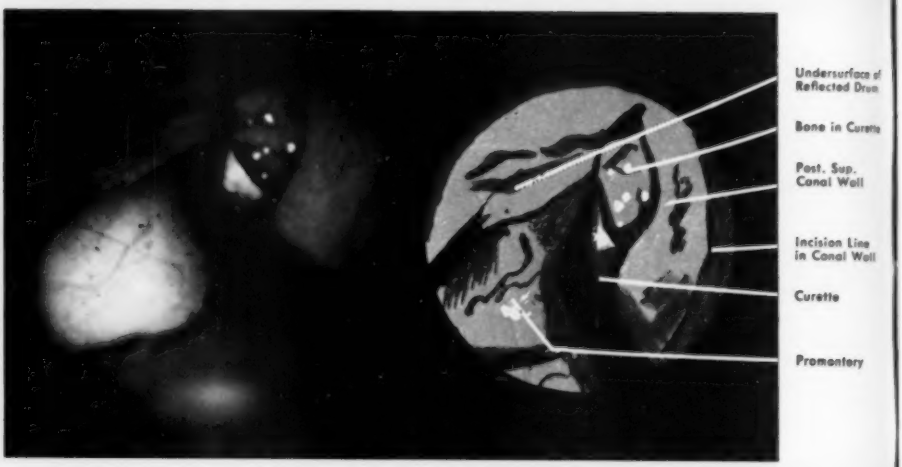


Figure 21.

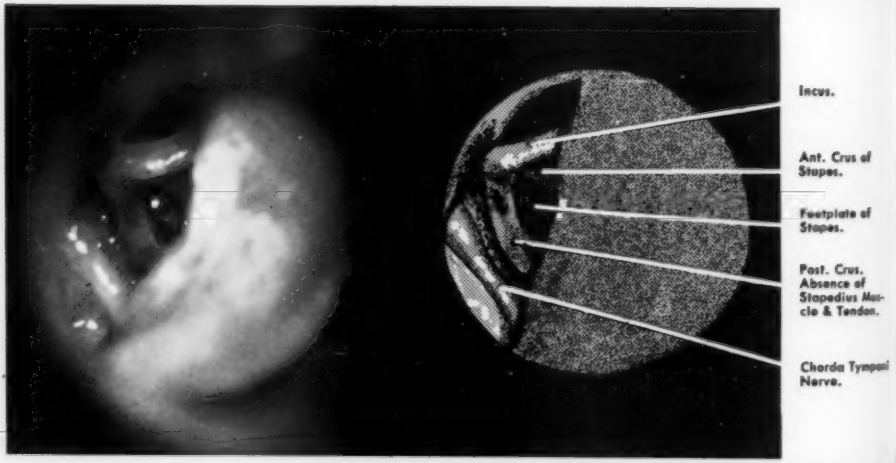


Figure 22.

Figure 21

This is a photograph of the very simple procedure of obtaining a large curette full of bone from the edge of the posterior superior canal wall. The bone will then be used as an autogenous bone graft to restore continuity of the long process of the incus and the head of the stapes as has been here described and illustrated.

Figure 22

Photograph of the left middle ear presented to show total congenital absence of the tendon of the stapedius muscle and pyramidal eminence.

The author has now observed nine cases of this anomaly. There apparently has been no gross loss of hearing or adjustment as a result of this absence. This does not mean that this muscle is useless. Some of the finer aspects of human hearing may be lacking from the patient's experience.

This is used as a closely allied example. The incudostapedial joint may also be damaged and scarred and yet the hearing may be returned to near normal. This seemingly normal function in spite of fibrosis should not encourage license to do damage to this important structure.

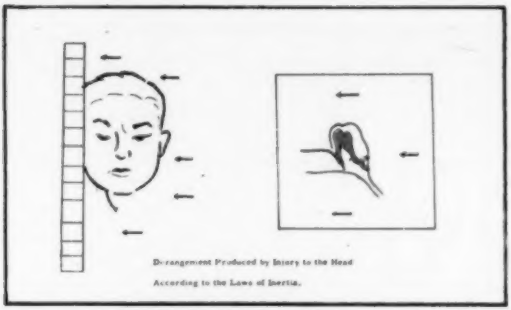


Figure 23.

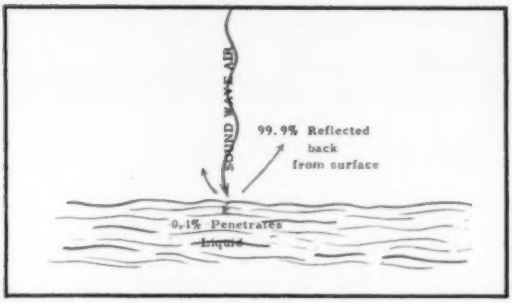


Figure 24

Figure 24.

Diagram demonstrating loss of transfer of sound from air into fluid as a result of impedence factors.

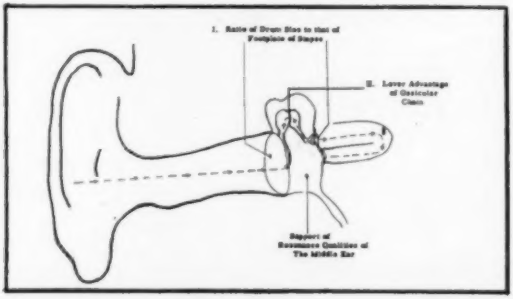


Figure 25

Figure 25.

Diagram demonstrating the important factors of the "transformer action" of the middle ear in the transfer of sound pressure energy from air to liquid.

ficiency. Investigation is now under way to ascertain the fate of bone grafts in the middle ear.

A reasonable inquirer would desire to know how the normal, gliding smooth surface of the joint could be scarred, fibrosed, or perhaps ankylosed, and still allow almost perfect sound transmission through it. This operative restoration of hearing with such a fibrotic joint may give us a much clearer concept of the function of this joint. It is true, that the ability of the joint to transmit sound does not seem to be lost with intense scarring and perhaps ankylosis, but sound transmission is only a part of the function of the incudostapedial joint. In fact, this joint seems to be more of a juncture along the pathway of sound transmission at which sound conditioning may occur.

Frequently, we have considered this joint as nothing more than something to connect one ossicle with another so that vibratory energy might pass. It is the smallest joint in the human body. This, however, points not to its insignificance, but clearly to delicacy and fineness of purpose. I have observed nine cases of total absence of the stapedius tendon, one of which is demonstrated in the photograph represented by Fig. 22. These patients do not complain of an inability to hear simply because the stapedius tendon is missing. This means that grossly they do not need a stapedius muscle to hear, but some of the finer aspects of human hearing may be lacking from their experience. The stapedius muscle and the incudostapedial joint apparently hold a togetherness in the conditioning of sound as it passes toward the cochlea.

Heretofore, we have had the opportunity to test only animals under experimental conditions, but now we may learn much regarding the physiology of the middle ear by a careful analysis of these human cases with isolated defects.

SIGNIFICANCE OF INCUDOSTAPEDIAL JOINT SEPARATION.

I. Significance of Incudostapedial Joint Separation Due to Surgical Procedures.

The previous discussion and information in the literature regarding an incudostapedial joint separation incident to

mastoid surgery speaks of its own significance without further elaboration.[27,31]

Trans-incus and stapedial head maneuvers are still being used effectively to mobilize the stapes, but extreme care should be exercised not to injure the incudostapedial articulation (see Fig. 6). A crushing fracture of the head of the stapes or lenticular process of the incus should be meticulously avoided, since these parts frequently necrose after fracture and produce pathologic separation of the two bones.

Any surgical maneuver in the middle ear can cause accidental interruption of the ossicular chain, but in no procedure should one deliberately separate the incudostapedial joint simply because the operator might erroneously consider it to be unimportant.

II. Significance of Incudostapedial Joint Separation Due to Inflammatory Necrosis.

Inflammatory necrosis of the long process of the incus may cause total incudostapedial joint separation (see Fig. 8). This will cause a near 60 db conductive loss of hearing.[13] This is very commonly encountered in the tympanoplastic repair of lesions produced by an old healed inflammatory process. In most tympanoplasties, it is, therefore, important that the incudostapedial joint and the footplate of the stapes be exposed for microscopic functional examination. This can be easily accomplished through an ordinary incision for a mobilization of the stapes. Necrosis of the long process of the incus may be found in the presence of a normal tympanic membrane and without other significant damage of the ear.[13]

III. Significance of Incudostapedial Joint Separation Due to Head Injury.

Evidence found in some of the cases presented here adds information to our knowledge regarding the effects of injuries of the head on the ability to hear. Conductive deafness, due to ossicular chain damage, is a strong possibility in these cases (see Figs. 9, 12, 15; Audiogram I). We now know that severe head injuries may produce:

1. Cochlear damage alone as has been previously well emphasized.[6,7,10,11,12,17,18,19,20,24,25,30]

2. Conductive deafness alone, as is seen in these cases of incudostapedial joint separation.[13]

3. A mixture of both conductive and perceptive deafness (Audiogram III).

Many of these cases will present medicolegal problems involving a personal or an industrial liability claim (see Fig. 13). Proper interpretation of test results combined with well planned surgical treatment will enable us more justly to assess and correct losses in these cases. A loss of hearing immediately following a head injury, whether it be conductive or perceptive, must now be considered to be the result of that incident. Before final disposition and after a reasonable time, exploratory tympanotomy should be done in *all* cases of otherwise unexplained conductive deafness, including those following injury of the head (see Figs. 9, 12, 15).

IV. Physiologic Significance of Incudostapedial Joint Separation.

Many exceptionally brilliant thoughts have come to us by past publications that we may be enlightened as to the function of the foremost of all sound receivers—the human ear.[3,21,23,35,36,37,38] In spite of the considerable amount of knowledge on this subject, otologists do not always understand or use this knowledge which embraces certain basic principles. Despite functional significance, many of the present surgical procedures of the middle ear produce ossicular chain separation either deliberately or by accident.

A brief statement as to the general function of the middle ear is that it receives sound vibration in air and transforms it to become sound vibration in fluid. How this is accomplished has been the subject of much speculation in the past. All authorities, however, generally agree, because of physical facts, that a transfer of sound vibration by air cannot be made directly into fluid without a marked loss of energy (see Fig. 24). Only 0.1 per cent penetrates the fluid (see Fig. 24); 99.9 per cent of this vibrating energy is lost or

reflected back at the fluid surface. This means acoustically that the sound in the fluid would be about 30 db less intense than in the air.[21] This can be easily calculated by consideration of the relative impedance values of each substance.

The primary function of the middle ear is that of a transformer mechanism which transfers the energy from the medium of air to the medium of fluid without loss of loudness.

As we have observed, the transfer of air to fluid normally presents a near 30 db loss (see Fig. 25). The middle ear overcomes this deficit in several ways. The following are some of those methods used:

1. The vibratory force of sound is exerted on a large tympanic membrane and is collected and transferred via the ossicular chain and concentrated on the smaller area of the footplate of the stapes. This represents a gain of ratio between the two active areas of approximately 28 to 1.[21]

2. The ossicles are pivoted in a manner which provides a mechanical lever advantage of 1.3 to 1.5:1. This adds an additional 1 to 2 db.

3. As a transformer, the middle ear has certain qualities of resonance of its own which exert an influence on the vibrating system of the ossicles to produce either a reinforcement or dampening of the vibratory energy passing through it. This results in a most complicated combination since this resonant effect varies according to frequency and intensity. Although these associations are complex, one may state definitely that the middle ear is amazingly well suited, in its own resonant properties, to perform this purpose of sound conduction and sound conditioning.

The preponderance of evidence supports the theory that sound is collected by the tympanic membrane and transferred via the ossicular chain to the footplate of the stapes whereupon entry is made into the inner ear.[21,37] From time to time, there have been suggested theories which deny the pre-eminence of the drum and ossicular chain as the primary conductive route into the inner ear. One extreme is the theory of hearing in reverse which proposes that sound penetrates the drum and

goes through the round window into the cochlea. The ossicles and drum act as a protector. These thoughts all lead to the erroneous teaching that removal of certain sections of the ossicular chain is unimportant. It has been stated authoritatively that in the normal ear the vibrations entering the drum ossicular route have at least as much as 1000 times the energy of those entering by the round window.[21,37]

Actually, to prove again the importance of the ossicular chain in sound conduction, one need only review the numerous animal experiments and physical data which have accumulated. I will mention three uniquely different experimental approaches to the analysis of the conductive mechanism.

1. Many authorities have carried out carefully controlled experiments on animals.[21,37,38,39,40] The incudostapedial joint was separated without disturbing the remainder of the middle ear mechanism. There was always produced a profound loss of approximately 60 db in the middle range of hearing when the incudostapedial joint was separated. The hearing levels in these cases were tested by using measurements from cochlear electrical potentials.

2. Evidence under another approach was that done by a physicist who utilized mechanical and physical laws to draw up a mathematical model of the ear.[23] From it, rather precise and amazingly accurate evaluations of the relative values of the components of the ossicular chain can be calculated. This mathematical formulation places a value of approximately 60 db upon a separation of the incudostapedial joint.

3. Work done by another authority on the condensor microphones has given us a technique that makes possible measurements by direct observation of movements of the vibration of the footplate of the stapes on the cochlear side.[3] One can also measure the vibrations of the tympanic membrane on its external side. In this microphonic device, one plate of the condensor is attached to the vibrating stapes, or tympanic membrane, while the other plate is fixed. The vibration alters the distance between the two plates and thereby produces a change in the copacetance of the condensor. Thus, the amplitude of the vibration can be easily measured. The

effect of ossicular chain interruption on these amplitudes of vibration can be determined.

These are only a few of the experimental methods by which the incudostapedial joint may be studied.

From a practical viewpoint, information derived from patients presented here will add clinical evidence to that already obtained in previously experimental studies.

Until recently, physiologic study of the incudostapedial joint has been confined to animal experimentation. Patients presented here (see Figs. 6, 7, 9, 10, 11, 12, 13, 14, 15, 16) represent human cases in which the importance of the incudostapedial joint can be studied directly. This *in vivo* human evidence either supports or denies certain assumptions concerning the transmission of sound vibrations via the ossicular chain. This evidence is well documented by actual photographs of each case, along with records of functional results before and after repair of the joint separation.

Fig. 10 is that of a classic incudostapedial joint separation.[13] This patient had a unilateral purely conductive loss of 67 db for pure tone in the speech range and a 62 db speech threshold. We must assume all of this loss to be purely and completely due to incudostapedial joint separation. No other abnormalities of the external or middle ear could be found. The tympanic membrane was normal and the malleus and incus moved easily. The round window was normal. The stapes was easily seen in its entirety and its footplate could be observed to move microscopically normally. This cochlea had the opportunity to receive sound unimpeded through either the round window or the oval window. There were no mechanical barriers at either window. Both avenues of entry to the cochlea were perfectly normal, and all else concerning this ear was apparently in order; yet, there was a loss of 67 db in the speech range (see Audiogram I).

The only abnormality in this ear was a wide separation of the incudostapedial joint. An incudostapedial joint separation, therefore, may categorically be said to cause a near 60 db conductive loss of hearing in the human ear.

To emphasize this firm statement, we may observe the restoration of hearing in this patient resulting from a repair of the incudostapedial joint (see Fig. 11, Audiogram II).

Repair of this joint alone was responsible for a hearing improvement. Nothing else was done to the stapes or its footplate or any other portion of the ossicular chain or tympanic membrane which might have accounted for this improvement. The gain must presumably be due only to the re-establishment of contact between the incus and stapes.

In summary, by the use of several components, the middle ear acts as a transformer which overcomes about 30 db which otherwise would be lost in the transfer of sound energy from air to fluid. If there is a gap in the chain across the transformer, such as is represented by the separated incudostapedial joint, not only the transforming action of 30 db is lost, but a short circuit of another 30 db is imposed. This makes a total of a 60 db loss of hearing produced by incudostapedial joint separation.

It is immmediately apparent that in this case it would be better to remove the transformer entirely and suffer only the 30 db loss in the transfer of sound from air to fluid. Indeed, experimentally in those cases where the incudostapedial joint was separated, the hearing could always be improved by 15 to 30 db if the tympanic membrane and outer ossicles were removed. This is the same as a system without a transformer.

This then brings us rapidly to the conclusion that if there is an incudostapedial separation, the tympanic membrane and ossicles are not only useless, but, in addition, act as a soundproofing shield which absorbs and reflects vibratory energy; therefore, in a given case, if there is to be no provision for a solid ossicular chain between the tympanic membrane and footplate of the stapes, it is only logical that the tympanic membrane and outer ossicles be entirely removed! This, of course, is unreasonable and is mentioned only to emphasize the importance of maintaining the integrity of the ossicular chain. It also points out that one must not remove an ossicle or disrupt a joint between two ossicles without making provision

for the restoration of a solid pathway over which sound may travel from the tympanic membrane to the footplate of the stapes.

Increased knowledge of tympanic physiology is accumulating rapidly. Microscopic exploration of all areas of the middle ear has become extremely common in recent years. Many of the surgical procedures cannot be routinely followed in a step-like fashion. Due to the tremendous number of anatomical and pathological variations encountered in this area, operations must be individualized to meet the needs of each patient. This is axiomatic in all tympanic surgery. To repair the conductive mechanisms of the middle ear, therefore, one needs not an organized procedure in mind, but an understanding of the purposeful design of each component part of the mechanism. Complete understanding may not be ours yet, for now we know in part; nevertheless, we see enough grandeur in what we do comprehend that we should never be tempted into disrespect for any portion of this beautifully created organ.

SUMMARY.

The etiology of incudostapedial joint separation may be on an embryologic, surgical, inflammatory, or traumatic basis.

Repair of this separation may be accomplished by stapediopexy, repositioning, or autogenous bone graft. Some suggestions in technique have been made.

In the study of this joint separation, one is impressed by its frequency, its profound effect on the hearing, its physiologic implications related to all middle ear surgery, and its relation to a wide range of causative factors.

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VETERANS OF FOREIGN WARS HONOR DR. ALBERT P. SELTZER.

Dr. Albert P. Seltzer was selected and honored by the Veterans of Foreign Wars as the Man of the Year on May 20, 1959.

COCHLEAR MICROPHONICS IN MAN.

A Preliminary Report.*†

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In 1930 Wever and Bray[6] first demonstrated the cochlear microphonic in cats. This phenomenon has been observed repeatedly in many species of animals, but there has been difficulty in recording the phenomenon in man. One of the earliest attempts was made by Fromm and his associates in 1935.[2] They were able to record only a small response in two cases. They used headphones to record the responses, and so no graphic records were obtained. In 1939 Andreev[1] and his group attempted to carry out further studies of the cochlear microphonic in man. Andreev was able to demonstrate the cochlear microphonic on the cathode ray oscilloscope with a maximum amplitude of 26 microvolts in one case, and a typical response of one to four microvolts. His larger responses were at 200 cps. Some of the intensities of sound used were noted to be painful. In 1941 Perlman and Case[5] published the first graphic record of the human cochlear microphonic. This response was barely above the noise level of the amplifier. Most of their records were made on unanesthetized subjects, by introducing the electrode through a perforation of the tympanic membrane. In 1947 and 1950 Lempert[3,4] and his associates reported further attempts to record this phenomenon. In 13 of 32 cases they obtained some response which was typically of low amplitude. In none of the studies reported

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were cochlear microphonics obtained that were comparable in amplitude or clarity to those obtained in working with experimental animals.

After review of the foregoing information, it was decided that another attempt should be made to obtain human cochlear potentials which could be recorded at a level, and of a purity, to permit more extensive studies of human cochlear function. These studies were carried out in the operating rooms of the Department of Otolaryngology, using the equipment, and under the supervision, of the Neurophysiological Laboratory of the Department of Otolaryngology.

METHOD.

Recordings were made in a general-type operating room under sterile conditions. The room was quiet but not soundproof. The subject was not shielded. The wires from the electrodes were shielded, and as many as possible of the AC circuits in the operating room were turned off during the recording period. There was no interference with the physiological signal.

All stimuli were delivered to the exposed middle ear at the operating table. Stimuli were: *1.* human whistle; *2.* handclaps; *3.* human voice; *4.* tuning forks; *5.* tones from an audio-oscillator. The effective stimuli were of moderate intensity, far below tactile level.

The recording electrodes in Cases H-1, H-2, and H-3 were made of 26-gauge Teflon-coated stainless steel wire with a small ball tip. In Case H-4 a similar type of electrode was fashioned from platinum. An indifferent electrode of the same material as that of the recording electrode was placed in the temporal muscle through a small stab-wound. The subject was grounded by means of an electrocardiographic electrode applied to the shoulder nearest to the ear being tested. The potential difference between the indifferent and the recording electrode was recorded. The responses were amplified by a Tektronix 122 amplifier, which was connected to a Dumont 403-R cathode ray oscilloscope. Graphic records of the responses were made with a Dumont cathode ray oscilloscope

Polaroid camera. The traces were synchronized with the response. The time-base and the amplitude are indicated in the lower right-hand corner of each figure.

OPERATIVE PROCEDURES.

Case H-1. B.T., age 12, white female, No. 839291. Past history of chronic discharge from the right ear. Clinical diagnosis: chronic tympano-

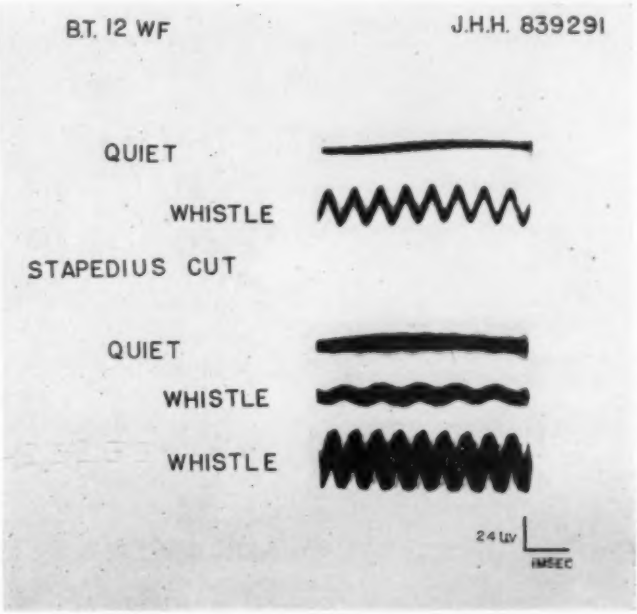

Fig. 1. Case H-1. Response at the round window membrane to human whistle before and after section of the stapedius tendon.

mastoiditis with secondary cholesteatoma arising from a marginal posterior-superior perforation in the tympanic membrane. A right endaural mastoidectomy was performed. The cholesteatoma was found to extend into the aditus ad antrum and to have destroyed part of the body and the long crus of the incus. Following the removal of the cholesteatoma a membrane of scar tissue could be seen, external to the round window membrane. After a small portion of the lateral margin of the round window niche was curetted away and the pseudo-membrane was removed, a stainless steel electrode was placed on this membrane. An excellent exposure of the round window membrane was obtained. After a series

of stimuli had been recorded the stapedius tendon was sectioned and further recordings were made. Representative responses are shown in Fig. 1. Following the recordings, a Type III-B tympanoplasty was performed. Preoperatively the patient had a 20 to 30 db loss in the lower tone ranges, and a 20 to 25 db loss for high tones. Postoperatively, the hearing has not been tested.

Case H-2. E.H., age 72, white female, No. 840760. This patient had a history of vertigo, staggering to the left, and discharge from the right ear. Clinical diagnosis: chronic tympano-mastoiditis with secondary

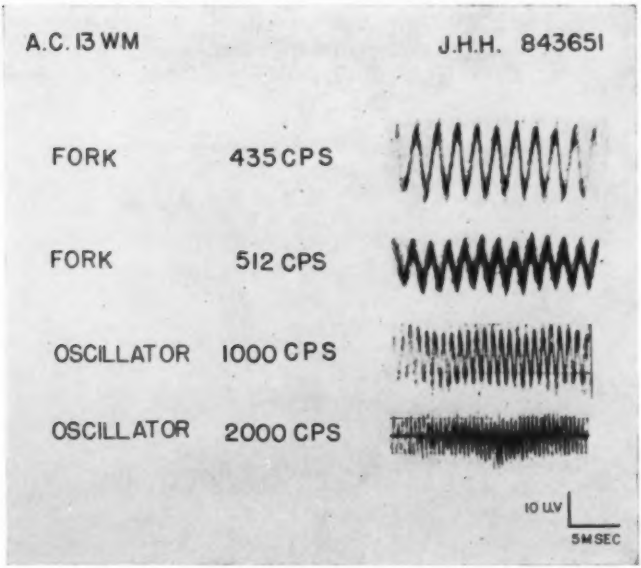

Fig. 2. Case H-4. Response at the round window membrane to stimulation with various tuning forks and tones from an audio-oscillator.

cholesteatoma arising from a marginal posterior-superior perforation in the tympanic membrane and labyrinth fistula. The right middle ear was exposed by an endaural approach. The cholesteatoma was found to occupy the attic and to extend through the aditus into the anterior portion of the antrum. It had fenestrated the ampullar end of the lateral semicircular canal. The stapes was embedded in thick scar tissue. Some granulations were cleared away from the round window niche. The round window membrane could not be visualized. The stainless steel recording electrode was placed in the niche and minimal responses to sound stimuli were noted. The electrode was shifted to obtain better approximation to the round window membrane. Two or three drops of clear fluid welled out of the round window niche; the round window membrane was thought to have been perforated. No further attempts were made to obtain re-

cordings. The surgical procedure was terminated by a modified III-B tympanoplasty. The patient had an uncomplicated postoperative course. Her clinical symptoms improved. Preoperatively she had a 40 db loss in the right ear; postoperatively it was slightly improved.

Case H-3. M.R., age 46, colored female, No. 613659. This patient had a history of a right radical mastoidectomy in 1952, with subsequent chronic purulent discharge from the operative cavity. When the cavity was revised the stapedial footplate was found to be embedded in granulations and scar tissue. The crura had been destroyed. The round window niche was filled with scar and granulation tissue. After this was removed a portion of the lateral margin of the niche was curetted away in order to expose the round window membrane. In spite of this, good visualization of the membrane was never obtained. A stainless steel electrode was placed in the scar tissue in the niche with the ball tip pointed upward toward the membrane of the round window niche. Using sound stimuli of human whistle and tuning fork, only minimal responses could be elicited. Following the above, a Type IV-A tympanoplasty was done. Postoperatively, the patient had some nystagmus and vertigo; this cleared up in 48 hours. A postoperative audiogram showed an average gain of 20 db in acuity.

Case H-4. A.C., age 14, white male, No. 843651. This patient had a history of recurrent otitis media and a persistent, small central perforation in the posterior-superior quandrant of the right tympanic membrane. The middle ear was exposed by creating a posterior tympano-meatal flap, and by elevating the posterior portion of the fibrous annulus and folding the tympanic membrane forward. The round window membrane was easily visualized. The inferior portion was covered with a small pseudomembrane. A platinum electrode was placed on the membrane. Excellent cochlear microphonics were obtained in response to sound stimuli (see Fig. 2). The recordings were followed by a transmeatal closure of the central perforation of the tympanic membrane. The patient's postoperative course was uneventful. Postoperatively, the hearing was improved.

FINDINGS.

Significant cochlear microphonics were obtained in two of the four cases. In both of these cases the recording electrodes could be placed directly on a normal appearing round window membrane. If the electrode was shifted to the bone surrounding the round window, the amplitude of the microphonic was greatly reduced. In the two successful cases low intensity sounds such as whispered words, or spoken voice in the operating room, could be readily visualized on the cathode ray oscilloscope. In these cases the human cochlear microphonic was similar to that obtained from a cat.

In Case H-1 (Fig. 1), a 36-microvolt response was obtained from a high-pitched human whistle. Following section of the stapedius tendon, a whistle of approximately the same intensity gave a 42-microvolt response. A low-pitched whistle gave a 6-microvolt response following stapedius section.

In Case H-4 (Fig. 2), sound stimuli were presented by means of tuning forks, an audio-oscillator, and human whistle. The 435-cycle tuning fork elicited a 25-microvolt response. The 512-cycle tuning fork gave a 20-microvolt response. A 1000-cycle tone produced an 18-microvolt response. A 2000-cycle tone produced a 12-microvolt response.

We had no accurate measurement of the sound input. Some

estimate can be made from the fact that the human whistle was presented by the operator through a standard face-mask from approximately two inches. The tuning forks employed were steel; they were activated by striking them against the operator's elbow, which was padded by a gown. The tone from the audio-oscillator was delivered through a brass tube approximately 2 cm. in diameter, 25 cm. long, placed approximately two inches from the ear. At no time during the recording did the forks or the speaker come in contact with the subject. Without more accurate measurements of sound intensities, we cannot make generalizations regarding cochlear sensitivity.

In order to evaluate further the order of sound-pressures employed, a cat was anesthetized, the middle ear exposed through the bulla, and the platinum electrode used in H-4 was placed on the round window membrane. The tympanic membrane was partially excised and sound stimuli were presented to the cat's ear, using the same tuning forks and audio-oscillator. Comparable cochlear potentials were obtained to those seen in Cases H-1 and H-4. When the stapes was fixed with dental cement, no satisfactory microphonics could be recorded with these sound sources.

CONCLUSIONS.

1. Cochlear microphonics in man were obtained at a level which permits more extensive studies on human cochlear function.

2. Cochlear microphonics in man, as in the cat, would seem to be greatly influenced by the degree of fixation of the stapes and the patency of the round window.

3. To obtain satisfactory recordings of cochlear microphonics in man the recording electrode must have a good contact with the round window membrane.

SUMMARY.

During the course of four middle ear operations, attempts have been made to demonstrate the cochlear microphonic response in man. In two of the four cases responses were

obtained, and recorded, that are like those yielded by the ears of experimental animals.

ACKNOWLEDGEMENTS.

We thank Dr. W. B. Kouwenhoven for the use of some electronic equipment; Drs. Alderman, Nicol and Cherry for their assistance at the operations; and all of the operating room nurses and anesthetists who worked overtime.

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL.

The Gill Memorial Eye, Ear and Throat Hospital has just completed its Thirty-second Annual Spring Congress in Ophthalmology, Otolaryngology and allied specialties. The attendance was one of the largest in the history of the school with an attendance of 350 physicians and their wives. There were forty-two states, England, Canada and several foreign countries represented. There were twenty-one guest speakers, sixty lectures and closed circuit televised surgery during the five and one-half days of the Spring Congress. In 1960, the Thirty-third Annual Spring Congress will be held from April 4 through April 9.

TYMPANOPLASTY.*

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With the introduction of tympanoplasty by Wullstein and Zollner, great interest has centered on this phase of hearing restoration. As Frenckner[1] has pointed out, "the solution of the great problems demand good teamwork, and it is justifiable that all who are intimately occupied with this problem, should present their views as support to the others, and as elements in the teamwork encompassing the entire otologic world." The purpose of this presentation, therefore, is to discuss various phases of this surgery in the light of obtained results.

In considering this phase of auditory restorative surgery, we must alter our criteria, indications and mode of treatment. Where the patient retains adequate cochlear reserve, every effort should be made to restore the hearing acuity, and in the course of treatment, eliminate the underlying disease. This, of course, excludes such contra-indications as osteitis of the labyrinthine capsule and cholesteatoma of the inner ear. The conservative care, especially of cases with cholesteatoma or other dangerous pathology, should no longer be condoned.

Zollner,[15] Wullstein,[12] Frenckner,[1] and many other authors, have considered the one-stage vs. the two-stage operation. Where the patient is suffering from acute infection, with danger to the general well-being, or with complications involving the meninges, lateral sinus or facial nerve, the immediate situation should be relieved, preserving as many of the middle ear structures as possible. The infection can then be brought under relative control and the tympanoplasty procedure carried out as a second stage operation. A critical view should be extended to excessive radical surgery in acute situations. On the premise that operative measures remove

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the underlying pathology, all other tympanoplasty operations are carried out as one-stage procedures.

Obviously, every effort is exerted in the preoperative period to obtain the quiescent state. Exhaustive efforts are made to eliminate the aural infection, but a draining ear does not deter from proceeding with the surgery. Contributing nasal and pharyngeal disease is eliminated, and the general physical status is evaluated, and appropriate medical measures are instituted. Mastoid films are often informative. The hearing should be evaluated by pure tone audiometry and other methods to determine cochlear function. The status of the ossicles and round window can often be determined with prosthetic patching, the "acoustic probe" of Zollner, and with damping prosthesis over the round window. The function of the eustachian tube can be evaluated by inflation, autoinflation, the passage of dye solutions and the passage of contrast media with roentgenographic study.

Ascertaining the cochlear reserve is of utmost importance, but the other tests are less reliable, may produce a state of false security and distract from the execution of the exacting steps in producing satisfactory results. Regardless of the preoperative appearance of the tympanic membrane and the results of the examinations, the status of the ear cannot be adequately determined until the time of surgical intervention. A preoperative plan of procedure is determined, but in every case the surgical team and the patient should be prepared for the extension of the operation to any given type.

Many specific cases could be mentioned to exemplify this concept, but this should suffice. On one occasion, an individual was observed with small, dry central perforations bilaterally and no visible middle ear disease. The cochlear function was adequate, and patching prosthetics improved the hearing acuity. On each occasion, surgical intervention revealed cholesteatoma in the oval window niche, having destroyed the head and crura of the stapes, pressing on the medial surface of the tympanic membrane, and thus producing a "columellar effect" between the tympanic membrane and stapes footplate.

With regard to the type and source of the skin graft, a variation of opinion is prevalent. There is a great deal of discussion by the various authors (Pagett,[4] Guilford and Wright,[2] Wullstein,[12] Harris and Meadows,[3] House[5]), regarding the split thickness vs. the full thickness skin. Both the three-quarter split thickness and the full thickness grafts would provide the much-discussed superficial and deep vascular networks and the strength desired by Wullstein. The post-auricular skin has been used exclusively in all of our cases, being readily available, almost free of glands and hair, and its corium contains only a few elastic fibers so that it does not shrink nor curl.

The skin is made as thin as possible, and the edges are beveled. One may err in not thinning the graft adequately, fearing that the trauma may be detrimental. A great deal of pressure is exerted to force the excessive tissue between the blades of the scissors, and at no time has the failure been due to primary sloughing. Where the skin is to be reflected on the adjacent external auditory canal, small wedges are removed from the periphery to avoid wrinkles.

If the skin is obtained from elsewhere on the body, an endaural incision may be justifiable. Where the graft is obtained from the post-auricular area, a second incision in the ear is not within the bounds of good surgical judgment. When using the post-auricular approach, one should observe that the skin of the superior half of the post-auricular fold is tightly bound and not in excess; whereas, the skin of the inferior half of the fold is loosely bound and often hangs in folds behind the lobule; hence, a large "tear-drop" shaped incision is made, removing a large portion of the skin from the inferior three-fourth of the post-auricular fold. The skin incision can then be extended superiorly so as to gain adequate exposure. The perichondritis reported by Leland House[6] has not been encountered. When properly made, the incision affords excellent visualization of all areas, and the extensive incision of Herrmann, as used by Zollner,[14] is considered excessive and unnecessary.

Many otologists, especially in this country, prefer the transmeatal myringoplasty to the classical tympanoplasty Type I.

This procedure was undertaken more than 70 years ago by Berthold and Tangemann, but was later abandoned.[13] Wullstein has observed that the operation which solely closes the perforation, will often produce excellent initial hearing gain, with later deterioration due to scarring of old granulations. In the early days of development, myringoplasty was carried out by the European schools. On revision and re-evaluation of the unsuccessful cases, Kley[7] found pathology in one or more of the five control areas as described by Wullstein. On the basis of these findings, the tympanoplasty Type I has been used, and unsuspected pathology has been encountered in the majority of cases.

The various steps of the Type I tympanoplasty are well documented, but several minor factors warrant further discussion. The external auditory canal is enlarged to the extent that every portion of the annulus can be well visualized. There is no hesitancy in removing a large portion of the anterior canal wall, and no effort is made to preserve the overlying skin. The antral control should be as small as possible, just large enough to afford inspection of the mucosa and the antral contents. The literature is misleading in this respect, causing some workers to produce a very large antral opening, which increases the possibility of perforation of the overlying graft, producing secondary mastoiditis. Often, on thinning the posterior canal wall, a small cell is exposed. This may serve as an antral control, and further investigation may not be necessary. Solution is then passed through the perforation and suctioned from the antral opening, affording an impression of the status of the epitympanum and the patency of the additus ad antrum.

Early workers de-epithelialized a small area of the tympanic membrane adjacent to the perforation, but the present concensus is to denude the entire surface. Where the perforation approaches the margin, an adjacent two or three mm. of skin is removed from the external auditory canal. Zollner[16] often deflects the skin of the canal away from the annulus with the concept that the skin will later make better contact with the margins of the graft.

If the annular attachment of the tympanic membrane is

disturbed, the tension of the circular and radial fibers is lost, and removal of the epithelium becomes very difficult. The incision should extend only through the stratified squamous epithelium, so as to avoid damage to the underlying nutrient vessels. As Wright[11] has observed in his microscopic sections, the tissue removed consists of the outer stratified squamous epithelium and an equal thickness of underlying connective tissue. Because of this loose attachment to the radial fibers, the skin can easily be dissected in one sheet from the periphery to the handle of the malleus and the margins of the perforation. Along the handle of the malleus, the skin is firmly attached to the underlying periosteum; hence, great care must be taken in the dissection to avoid cellular remnants. The entire sheet is then removed with one mm. of the edge of the perforation.

Loose bands of radial fibers may simulate islands of epithelium, but these should not be disturbed. Frenckner[1] applies cautery with trichloracetic acid to suspected islands of epithelium, but this is considered unnecessary and can be destructive to the underlying graft bed.

The control of the upper middle ear by the Shrapnell's membrane approach and the lower middle ear by the fenestra, as described by Wullstein, has been replaced by reflecting the tympanic membrane, as in the stapes mobilization.

Consideration of middle ear pathology warrants a separate presentation, but since myringoplasty was mentioned, it may be well to discuss some of the conditions encountered in the Type I operation. When the tympanic membrane is reflected so that the medial surface of the perforation can be visualized, it is not infrequent to find that a column of diseased tissue extends from the perforation to the promontory and often spreads to encase part or all of the middle ear structures. This tissue can often be swept out as one sheet, to find that the underlying structures are covered with their own thin and healthy mucous membrane. Stratified squamous epithelium can migrate down this column to the middle ear structures; hence, its removal is of great importance. Fibrous bands and membranes often bind the ossicles and cover the round window niche. Purulent secretions, filling the antrum and mastoid,

were encountered in a clean, dry ear of long standing. Cholesteatoma in various degrees of destruction and in many unusual areas has been seen. On two occasions, ankylosis of the stapes was encountered with good results following mobilization.

In the more extensive procedures, where disease and cholesteatoma are present near the stapes and in the posterior portion of the middle ear, great care must be exerted in the proper dissection of the area. There is a consistent chain of perifacial cells emerging from the mesotympanum, lying lateral to the facial canal and inferior to the short process of the incus. There is a second group of cells inferior and medial to the above, lying anterior to the facial canal at the level of the hypotympanum. One should also bear in mind that the middle ear extends posteriorly, lying medial to the facial canal, forming the posterior tympanic recess.

Where the disease is present in these areas, the facial ridge is taken down to the facial canal in its descending portion in the region of the middle ear. The danger to the facial nerve in carrying out this dissection has been eliminated by the use of diamond burs. The cutting strokes should be light, "brush-like," and never with the exertion of pressure. A cholesteatoma involving this area may be removed *in toto*, but to circumvent the possibility of cellular remnants, the mucosa in its entirety is removed from the stapes or remaining portions thereof, and from the superior one-half of the middle ear, including the tissues of the posterior tympanic recess. In performing the Type IV operation, this dissection produces an area of clean, bare bone for the reception of the edges of the skin graft, the diseased tissue is removed and the secondary granulations, reported by Wullstein, have not been encountered.

Wullstein[12] has suggested the introduction of a plastic columella to convert the Type IV procedure into the Type III. Being skeptical of the introduction of foreign material into the middle ear, this procedure has not been carried out. The Type IV operation has afforded such astonishingly good results that further consideration should be given to the per-

forming of the Type IV, Cavum Minor operation, where the stapes is intact, as is advocated by Richtner.[10]

The Type V operation is preferably carried out in two stages, but the operation has been done in one stage with good results. If the operation is to be carried out in two stages, the fenestra is prepared down to the blue line. After the cavity is healed, a large skin flap is reflected from the medial wall of the created cavity. The bony cupola is then incised, lifted and removed, as was described by Lempert.[8]

Good eustachian tube function is an absolute prerequisite to all tympanoplasty procedures. For this reason, the eustachian tube is investigated in every occasion, regardless of the preoperative findings. Bony obstruction of the isthmus, requiring opening with a drill, has not been encountered. Where the tube is tight it is forcibly dilated with metal probes. In the majority of cases, the eustachian tube can be adequately enlarged by the passage of graduated plastic bougies from the middle ear to the nasopharynx. To maintain the patency of the eustachian tube during the healing period, Zollner[15] inserted an acrylic tube threaded with silk suture material. The many disadvantages led to multiple modifications until the present method was evolved. Polyethylene tubing is molded to conform exactly to the course from the round window niche to the vestibule of the nose. Prior to surgery, adequate lengths of p-50 and p-60 polyethylene tubing are selected. These are threaded with No. 26 stainless steel wires, which serve as mold forms, and the threaded tubes can be molded to the desired shapes. They are then immersed in boiling water for a moment followed by a cold water bath. The stainless steel wires can then be removed and the polyethylene tubes will maintain the desired configurations. A tube is selected, fastened to the end of a bougie and passed as one unit. This type of insert is advantageous in that the tube is neither felt nor is it visible. It is adequate in maintaining the patency of the tube, it allows for free drainage from the middle ear and, because of its conformity, it is self-retaining and cannot be extruded.

The gelatin hyaluronidase antibiotic mixture, as described by Kley[6] in his early experiments, is now used in all cases.

It is timportant that the gelatin pledgets be soaked for hours prior to instillation, so that all air bubbles have been disseminated, and the pledgets are in a gel state. Hyaluronidase is extremely important, as the only case in which poor pneumatization occurred was one in which only saline was used. The pledgets that are instilled should be extremely small, completely saturated and "floated" into position. Only enough is instilled to form a bed for the graft and excessive amounts avoided.

Prior to the instillation of the gelatin, all bone dust and blood clot should be removed. It is extremely important that every vestige of clot be removed from about the ossicles, the round window niche and from the external surface of the tympanic membrane. If complete hemostasis cannot be obtained by the application of topical adrenalin, a hypotensive agent may be employed. In applying the skin graft, firm pressure should be exerted to insure good apposition with the radial fibers and the annulus. One should be able to discern the underlying structures from the outer contour of the graft. In the Type IV, an opening is created in the graft slightly larger than the stapes footplate. The skin is firmly pressed onto the promontory and the inferior surface of the facial canal. The skin should be in direct apposition with the bone and the footplate visible in its entirety.

If the entire cavity cannot be covered with the skin taken from the post-auricular area, many workers will secure additional skin from other sites. The complete covering of the cavity with skin promotes early healing, but the additional time and surgical trauma involved in obtaining, thinning and placing of the skin outweigh this advantage. After the skin graft has been firmly pressed into position, the cavity is filled with additional soaked gelatin pledgets. A short length of one-half inch vaseline gauze is placed over the gelatin pledgets, carried through and packed into the external auditory meatus. The last maneuver serves as an aid in regaining or enlarging the size of the meatus.

The remaining skin overlying the mastoid process, upper cervical region and posterior surfaces of the lobule and auricle is widely undermined. The auricle is then drawn

posteriorly and sutured to the periosteum; this, too, aids in maintaining the size of the meatus. Retention sutures are insinuated from the under surface of the skin over the mastoid eminence and upper cervical regions to the periosteum at the original suture line. This draws the skin into position so as to regain the original post-aurical fold.

Innovations have been and are still being instituted in the postoperative care. Starting on the seventh postoperative day, the patient is advised to autoinflate until slight pressure is felt in the middle ear. On the fourteenth day, the polyethylene tube is removed with improved middle ear aeration. Late removal of the gelatin packing from the cavity has reduced the secondary infection and is conducive to rapid healing of the created cavity.

A compilation of the postoperative results will be deferred, as the surgical technique is still in the evolutionary state, and the elapsed postoperative time is too brief. In general, the results have been excellent, and this type of surgery lends much to the future.

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COURS DE TECHNIQUE AUDIOMETRIQUE.

The Faculty of Medicine, Paris France, will hold a Course in Audiometry, October 12-18, 1959, under the chairmanship of Prof. M. Aubry of the Clinic of Oto-Rhino-Laryngology. The tentative program includes lectures on the scientific fundamentals, the technique of audiometry, discussions of the results and problems of interpretation, and round table discussions. Professor Aubry will be assisted by MM. les Professeurs Agreges J. J. Debain and R. Maduro; les Doctears P. Aboulker, J. Bouche, P. Clerc, H. Henrot, R. Maspetion, J. Pialoux, J. Bouchet, M. Burgeat, L. Chevance; P.-L. Klotz, P. Robert, B. Vallancien; Mme. S. Borel-Maisonny; M. le Professeur A. Didier; MM. R. Chocholle, J.-E. Fournier, R. Lehmann and A. Molles. The complete program will be ready in June, 1959. For further details write Secretariet: Service Oto-Rhino-Laryngologique Pavillon Isambert, Hospital Lariboisiere. 2 Rue Ambrose-Pare, Paris 10°), France.

TWO CASES OF PRIMARY LATENT CHOLESTEATOMA DIAGNOSED AND TREATED BY TYMPANOTOMY.

Discussion of Pathogenesis and Management of Cholesteatoma.*†

HERMAN I. LAFF, M.D.,

Denver, Colo.

The following two cases of primary cholesteatoma in children having well pneumatized temporal bones and without previous history or symptoms of otitis media seem worth reporting. Both were referred after routine school surveys had shown a unilateral hearing loss.

CASE REPORTS.

Case 1.—E.P., female, age 9, came for examination on Feb. 22, 1958, because a recent school test had shown a hearing loss in her right ear. Neither patient nor her parents had been aware of any previous ear trouble. She had had the usual childhood diseases and a tonsillectomy and adenoidectomy four years ago. She has not been a mouth breather.

Examination revealed an obviously healthy school girl with normal ear, nose and throat findings except those of the right ear. The right ear drum appeared opaque and thickened, and lacked mobility. Careful search failed to reveal evidence of perforation. The Rinné test was negative in the right ear and the Weber was referred to that side.

An audiogram showed a typical conductive hearing loss of about 40 db in the right ear in most frequencies, with a corresponding speech reception threshold of 40. The hearing in the left ear was normal. Politzerization did not improve hearing. Roentgenograms of the mastoids revealed well pneumatized temporal bones with some clouding of the cells on the right but with their architecture still intact (see Fig. 1).

A presumptive diagnosis of primary cholesteatoma was made. A stapes-type tympanotomy was performed on March 28, 1958. The entire middle ear was found filled with cholesteatomatous masses extending toward the antrum and eustachian tube. These were removed by the use of suction and forceps. The middle ear showed evidence of encroachment and pressure atrophy, including destruction of the long process of the incus. The intact stapes, which was covered with a thin layer of granulations, was mobile. No evidence of ear drum perforation could be found. The drum and its meatal cuff were replaced and invaginated onto the head of the stapes in an effort to produce a columella effect. An appreciable hearing gain was thus obtained (see Fig. 2).

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†From the University of Colorado Medical Center.

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The postoperative course was uneventful. The ear drum now appears more translucent and moves with use of the Siegle speculum.

Case 2.—M.B., male, age 7½, whose hearing in his right ear had been found defective in a school survey made two weeks previously, was first seen on Jan. 31, 1957. The parents and child had not been aware of any previous hearing loss or ear trouble. He had had the usual childhood diseases and respiratory infections, including hay fever in the summer months. Ordinarily he has not been a mouth breather; his tonsils and adenoids had not been removed.

Examination revealed a moderate amount of mucoid nasal secretion and somewhat pale and swollen turbinates. The antrums transilluminated well. The tonsils and adenoids were only moderately enlarged. The left ear drum appeared normal. The right was dull and immobile. The Rinné test was positive on the left and negative on the right. The Weber

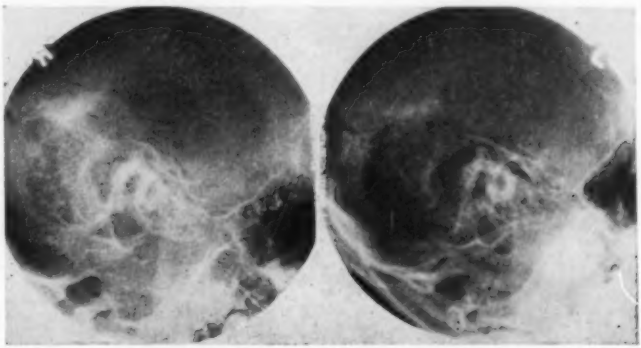

Fig. 1. Case 1.—Preoperative roentgenogram showing well pneumatized mastoids. There is clouding of cells on the right, but their architecture is still intact.

was lateralized to the right. An audiogram showed a conductive deficit in the right ear of between 35 and 40 db, with the lower tones relatively unaffected. A diagnosis of right chronic secretory otitis media was made and a tonsillectomy and adenoidectomy, together with myringotomy and aspiration advised. These were done on March 25, 1957. No fluid could be aspirated from the middle ear. Periodic hearing checks were advised, and when examined again on July 7, 1957 (3½ months later), the findings now suggested the presence of a primary cholesteatoma. X-ray of the mastoids revealed good pneumatization and cell clarity on both sides (see Fig. 3).

A stapes-type tympanotomy was advised and performed August 8, 1958. The findings were quite identical to those described in the previous case. The long process of the incus, however, was found to be less eroded. When some adherent cholesteatoma fragments in the hypotympanum were grasped, the ear drum was inadvertently torn. A piece of gelfoam saturated with Neo-cortef solution was then placed in the hypotympanum to hold the torn edges of the drum in closer approximation. A myringo-stapediopexy was attempted, utilizing the untorn portion of the drum.

It is now healed, shows areas of translucency and is fairly mobile. An audiogram taken on October 9, 1958, showed moderate hearing improvement (see Fig. 4).

DISCUSSION.

Before embarking onto discussion, a word about terminology and the confusing classifications of cholesteatoma in general may help toward a better orientation of just what we mean by its primary type.

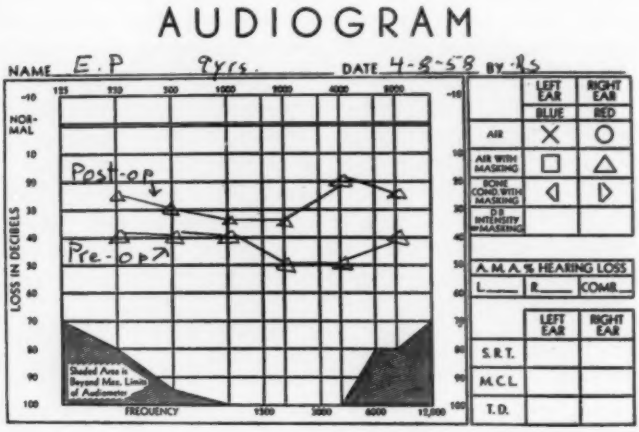

Fig. 2. Case 1.—Pre- and post-operative audiogram showing appreciable hearing gain following removal of cholesteatoma and myringo-stapediopexy.

Dr. Shirley H. Baron recently emphasized that true cholesteatoma, or cholesteatoma verum—usually a neurosurgical problem—should not be confused with the primary type. Texts have divided pseudocholesteatoma, which interests us as otologists, into primary and secondary, and then proceed to describe the common attic form with manifest perforations into Shrapnell's membrane as the primary type. Recently the same term has also been used for cases without perforation, such as I have described. Perhaps by adding a descriptive word such as *masked silent* or *latent*, to primary cholesteatoma, preferably the latter, there would be less confusion.

That the enigmas concerning the pathogenesis of cholesteatoma and the related processes of temporal bone pneumatization still exist, was amply demonstrated by the spirited discussion they engendered at the recent International Congress of Otolaryngology at Washington. As Rüedi[1] so aptly stated: "An exact knowledge of the pathogenesis of cholesteatoma is essential for the successful application of our new surgical techniques."

It seems that Witmaack's teaching that otitis media of infancy by inhibiting pneumatization predisposes to chronic

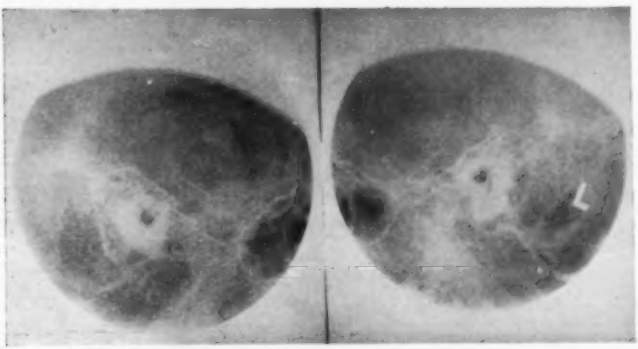

Fig. 3. Case 2.—Preoperative roentgenogram showing almost equal pneumatization and cell clarity of both mastoids.

infections of the temporal bone, is still being challenged. Rüedi prefers to believe that the relative lack of cells and sclerosis of the mastoid are not a cause, but a result of the chronic suppuration usually starting in early infancy.

Diamant's[3] studies have impressed him with the hereditary and prenatal factors in pneumatization.

Tumarkin[4] believes that frustration of pneumatization is dependent on a mechanical intratympanic vacuum created by continuous tubal closure, the result of chronic nasopharyngitis and adenoid hyperplasia in childhood.

The view that implanted embryonic cell rests in the attic

may form epidermoid cysts has also been proposed. The occasional true cholesteatoma occurring sporadically in the diploe of the cranial bones does seem to originate in this manner. Very few, however, now strongly support this theory for the origin of the common type of attic cholesteatoma.

Bezold's hypothesis that chronic tubal obstruction may lead to collapse and rupture of Shrapnell's membrane, followed by

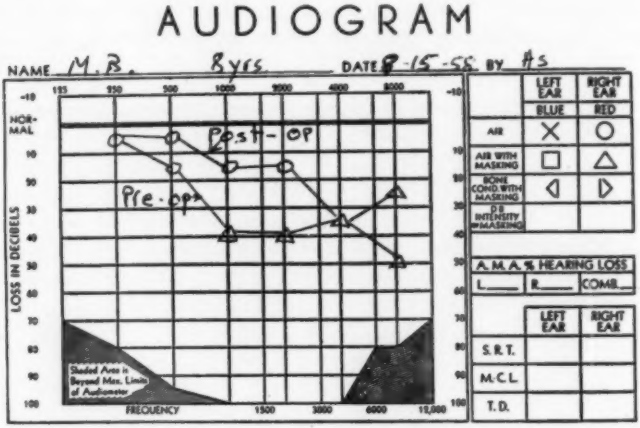

Fig. 4. Case 2.—Pre- and post-operative audiogram showing moderate hearing gain following removal of cholesteatoma and myringo-stapediopexy.

superimposed infection and otorrhea has also received varying acceptance.

Tumarkin's contention that the cuboidal epithelium of the attic could change to squamous type by an infectious metaplasia, was attractive in explaining primary cholesteatoma. He now no longer defends this view because of recent experimental evidence to the contrary.[4]

Clinical and pathological observation over the years have amply confirmed the belief that squamous epithelium from the external auditory meatus or tympanic membrane can migrate into the tympanum through marginal perforations, the result of necrotizing otitis media. Seldom, however, have

otologists actually seen the small marginal perforations originating and developing in Shrapnell's membrane during acute otitis media. This "missing link" has naturally cast some doubt on the validity of the classical immigration theory. Nevertheless, Rüedi[2] has histologic sections of embryos and young infants which show more than normal accumulation of hyperkeratotic epidermis of the upper drum margin. He believes that, as a result of recurrent low grade inflammation of the tympanum in childhood, basal cells of the intact pars flaccida "can multiply and grow into the submucous connective tissue filling the incompletely pneumatized attic, or into newly formed granulation tissue, where they continue to grow into a cholesteatoma." Thus, although having formed behind an intact tympanic membrane, the cholesteatoma usually perforates it early, giving rise to a small upper marginal perforation.

Recent experiments on guinea pigs tend to confirm this view. Friedmann[5] placed pathogenic bacteria into their middle ears and was able to produce perforations of their ear drums followed by typical cholesteatoma formation. Rüedi[1] has succeeded in producing cholesteatoma in guinea pigs behind their intact tympanic membranes (which interests us especially) by use of mild irritants (mixture of talc and fibrin). In man, however, histologic proof of primary cholesteatoma formation behind a still intact ear drum has apparently not been reported. Likewise, relatively few primary cases have been found clinically. Day,[6] House,[7] and DeWeese[8] each have recently reported such a case in adults.

Summarizing, no matter what theory of origin the nonpartisan observer leans toward, each theory presupposes that inflammation still is the all-important ingredient needed to activate attic cholesteatosis.

For a better understanding of the primary type, just what happens, especially in the early phase of a developing cholesteatoma and its matrix can be very informative.

The matrix, according to Rüedi,[1,2] manifests infiltrative qualities early in its efforts to seek out and replace the hyperplastic connective tissue remnants in the middle ear cleft.

Simultaneously, osteogenesis is also stimulated. When submucosal tissue has been replaced these early activities cease. The cholesteatoma then grows and expands by desquamation and infection at the expense of the surrounding bony walls, leading to complications all too familiar.

Primary latent cholesteatoma, it would seem, could result from disturbances of its early, typical growth pattern. Per-

Fig. 5. Cholesterin granuloma from a case of chronic mastoiditis showing granulation tissue containing cholesterin clefts, foreign body giant cells and histiocytes.

haps the ingrowing epidermis from the intact tympanic membrane becomes primarily attracted towards the newly formed granulations of the ossicles and tympanum. This could be encouraged by abnormal disposition of connective tissue or presence of strategically placed limiting bands and spaces in the attic. Thus Shrapnell's membrane fails to perforate.

Since cholesteatoma generally starts insidiously its possible association with the currently rampant masked forms of otitis and mastoiditis has been brought to the fore. Although in

the majority of the latter, the usual forms of non-specific inflammation have been found, in some there have been uncovered groups of cells under the cortex filled with blackish material and granulations full of foreign-body giant cells, histiocytes and cholesterin clefts. The latter represent dissolved cholesterin crystals (see Fig. 5). Some English authors[9,10] have looked upon these so-called cholesterin granulomas as precursors of attic cholesteatomas. They have been found in mastoids with and without attic cholesteatoma. As a rule, when both were present they were often remote from one another.

The once rare but now more commonly found "blue drum" syndrome or hemotympanum has also been included in the picture of cholesterin granuloma. The mastoid cells contain similar vascular granulations and slimy fluid discolored by hemosiderin. Incidentally, these must be thoroughly removed surgically for effective cure.

Whether cholesterin granuloma and attic cholesteatosis have a cause and effect relationship apparently has not yet been satisfactorily demonstrated. The finding of cholesterin clefts in granulation tissue, pathologists tell us, is a common foreign body reaction and is not confined to the temporal bone. Matrix in close proximity to the cholesterin granuloma has not been described. Similar granulomas have been found in pseudocysts developing in old radical and fenestration cavities. In my experience cholesteatomas have not resulted from these granulomas.

For the present it would seem well to keep an open mind toward these newer concepts. This may imply earlier surgical management of latent middle ear and mastoid infection, including those occurring in cellular temporal bones. Thus, Birrell,[9] of Edinburgh, found that close to 50 per cent of his cases of attic cholesteatosis in children occurred in pneumatized mastoids.

Today more "silent" attic granulations and perforations are being seen in children than say 15 or 20 years ago. One naturally wonders if this could be tied to the present increased incidence of fluid ears. Thus the removal of a speck of ceru-

men adjacent to Shrapnell's membrane will often disclose a small attic granulation and perforation leading into a cholesteatoma pocket. Often there had been no history of previous drainage from such an ear. In some, treatment for secretory otitis by myringotomy and aspiration had produced no improvement. One can assume that such cases in essence were "primary" cholesteatomas for months or years, until break through revealed their true diagnosis. In the two cases reported, the course was quite similar except that neither drum had perforated.

When cholesteatoma develops in sclerotic mastoids it tends to be confined to the attic and antrum areas by the hemming in effect of the dense bone; it also tends to become encapsulated by its matrix and gives the appearance of a cyst. This is a common finding, and the surgeon who chooses to preserve the matrix of such a cyst would appear to be on fairly safe ground. Where cholesteatoma develops in a diploic mastoid, one can expect to find finger-like extensions of matrix penetrating into deep-lying cells. These obviously should be meticulously sought for and removed.

What changes a growing cholesteatoma will produce in a well pneumatized mastoid, may be inferred from findings of previous radical mastoid procedures. Thus it could destroy the thin cellular partitions and occupy almost the entire mastoid process—a not uncommon finding (how this could have happened in a relatively short time in a child's sclerotic mastoid had been difficult to explain); or the cellular architecture could be crowded out by osteogenetic activity, thus converting it to a diploic type. This may be occurring in the case with cloudy cell outlines.

I chose to perform tympanotomy as a means of proving that cholesteatomas were present and of removing them, if possible. I suspect that they may recur as matrix may have been left behind.

It is significant that others are now recommending conservative measures in management of early cholesteatoma. Tumarkin[4] recently reported encouraging results with attic polypectomy and removal of epithelium from the borders of

the perforation, together with curettement of its bony margins. He used local anesthesia and magnification. In this way he could halt the epidermic invasion which had just begun at the surface.

Rüedi[2] proposed preventive antro-atticotomy with removal of connective tissue layers in the epitympanum in cases of recurrent infantile acute otitis media.

In well established types of cholesteatoma, however, most will agree with the latter's dictum that "the complete removal of the matrix, of all cell systems and all connective tissue contained therein is necessary."

SUMMARY.

Two cases of primary "silent" cholesteatomas in children having well pneumatized temporal bones have been presented. The latter finding helps to divert our thinking from an hitherto ingrained belief that cholesteatoma necessarily goes hand in hand with diploic or sclerotic mastoids.

Theories regarding pneumatization of the temporal bone and pathogenesis of cholesteatoma have been reviewed, especially as they relate to the primary type of cholesteatoma.

Disturbances in the early growth pattern of cholesteatoma and its matrix which may result in lack of tympanic perforation have been discussed.

The possible relations of cholesteatoma to the masked forms of otitis and mastoiditis, cholesterin granuloma and hemotympanum have been considered.

Exploratory tympanotomy as a means of diagnosis of primary latent cholesteatoma has been advanced. The definitive value of tympanotomy in treatment, however, is as yet unproven but is worth trying in selected cases.

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227 Sixteenth Street.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

The next joint annual meeting of the South Carolina Society of Ophthalmologists and Oto-laryngologists, and the North Carolina Eye, Ear, Nose, and Throat Society, will be held in Charleston, South Carolina, September 13-16, 1959. Headquarters will be the Francis Marion Hotel. Guest speakers will include Ophthalmologists: Dr. Caroll R. Mullen of Philadelphia, Pa.; Dr. G. Bonaccolto of New York, and Dr. Willis S. Knighton of New York; Oto-laryngologists: Dr. John Bordley of Baltimore, Md., Dr. Frederick R. Guilford of Houston, Tex., and Dr. Paul Holinger of Chicago, Ill.

For further details write Roderick Macdonald, M.D., Secretary and Treasurer, 330, East Main St., Rock Hill, S. Car.

OVAL WINDOW AND ROUND WINDOW SURGERY IN EXTENSIVE OTOSCLEROSIS.

A Preliminary Report.*†‡

WILLIAM F. HOUSE, D.D.S., M.D.,

Los Angeles, Calif.

The conquest of hearing loss due to otosclerosis is one of the most fascinating and unfinished parts of otology. The unsolved problems of otosclerosis call for continued search for medical and surgical methods of alleviating the handicap caused by this disease.

It is the purpose of this paper to present a method of opening the oval and round windows in cases of extensive otosclerosis.

OVAL WINDOW SURGICAL TECHNIQUES.

Stapes mobilization as proposed by Rosen[1] was a method of fracture through the otosclerotic focus of the oval window by pressure through the head and crura of the stapes.

Later footplate techniques developed by a number of surgeons have made it possible to mobilize the majority of otosclerotically fixed stapes; however, there still remain those patients with extensive otosclerosis in whom the thickness of the footplate is so great that it cannot be mobilized with any of the currently used needle, chisel or gouge techniques. The next logical step, therefore, seemed to be the development of a method of removing this thick otosclerotic bone.

The most practical method developed to date involves the use of diamond stones. These are used in a dental handpiece and are about the size of a No. 2 round bur. The cutting

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†From the Department of Otolaryngology, University of Southern California School of Medicine.

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end consists of a number of small diamond chips embedded in an almost pure nickel binder. Fig. 1 shows the application to the footplate.

The middle ear is exposed through the conventional stapes mobilization approach. The stapedius tendon is severed and the stapes head and crura removed. The thick otosclerotic

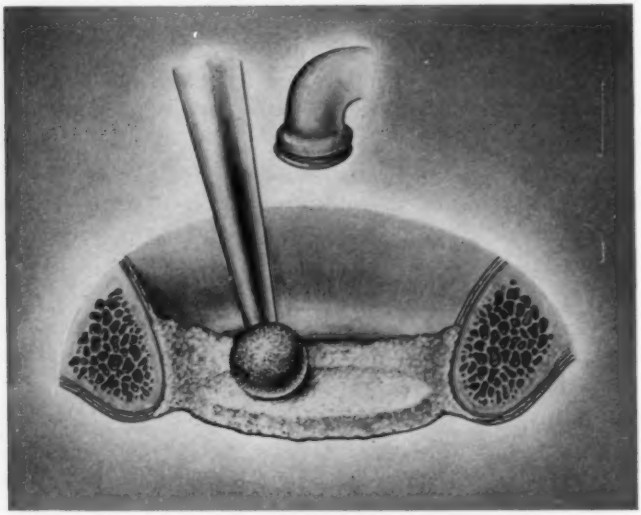


Fig. 1.

bone filling the oval window is then removed with a diamond stone. The stone must revolve slowly, in order to avoid heat. If the area becomes overheated the patient may experience a sensation of falling, due to utricular stimulation.

Gradually the oval window area is thinned so that it becomes bluish in appearance. A piece of No. 90 polyethylene tubing is then shaped and fitted, after the method of Shea[2] from the lenticular process of the incus to the footplate, as shown in Fig. 2.

When the strut is properly fitted and placed, the thinned

footplate is gently shattered by means of a chisel as shown in Fig. 3.

RESULTS OF OVAL WINDOW SURGERY USING DRILL.

To date we have operated upon six cases with the drill technique. Three have 75 per cent or more closure of the air-bone gap; two have 25 per cent closure, indicating a significant hearing gain, and one is worse by 12 db, as averaged in the

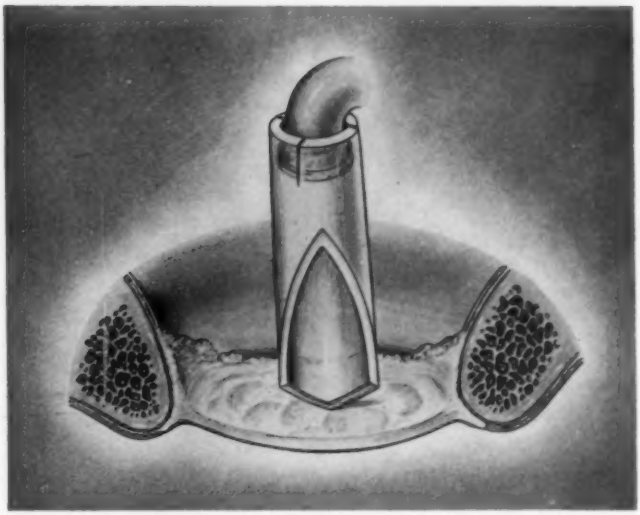


Fig. 2.

three speech frequencies. On this last case the hearing was improved for two weeks and then decreased suddenly, apparently due to a displacement of the polyethylene strut.

ROUND WINDOW OTOSCLEROSIS.

The round window is the second most frequent site of otosclerosis.[3] During stapes mobilization surgery this window should always be carefully examined. Frequently it is seen

to be partially occluded by otosclerosis. Apparently, however, unless the window is completely occluded none of the hearing loss can be attributed to this factor.[4] Nagar and Fraser[5] reported the histologic findings of six cases of total otosclerotic closure of both windows. All had almost total hearing loss of a perceptive type.

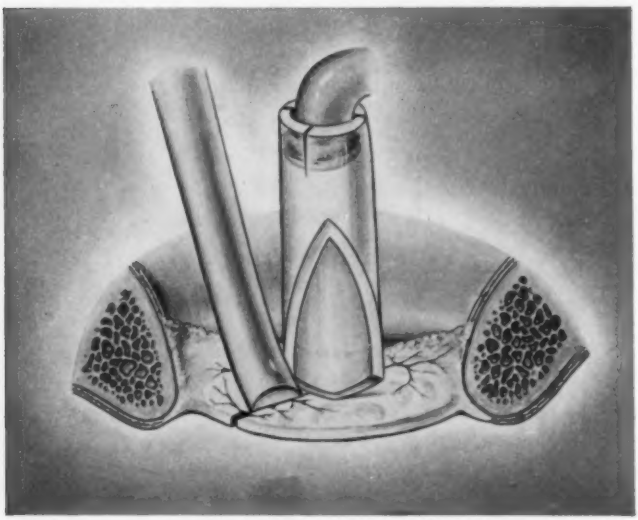


Fig. 3.

Since the diamond stone technique has made it possible to remove otosclerotic foci, we decided to see whether it was possible to improve hearing in far advanced otosclerosis by opening both the round and oval windows.

RESULTS OF OVAL AND ROUND WINDOW SURGERY USING DRILL.

To date we have results on six such cases four months or more following surgery. The following is a summary of three represenative cases of round and oval window otosclerosis.

Case 1. Mr. B., age 46, developed his hearing loss at the age of 18. A diagnosis of otosclerosis was established in our office in 1946. At that

time he had a conductive type hearing loss which was too far advanced for fenestration surgery.

In February, 1958, he was again examined and at that time responded only to bone conduction at 55 db at the 500 ferquency in each ear. No other bone or air conduction responses were obtained at the limit of the audiometer. The patient was wearing a bone conduction aid, but was unable to understand any speech with the instrument.

X-rays made by Dr. Compere revealed extensive otosclerosis of both otic capsules.

In June, 1958, through an endaural incision, the left middle ear was exposed. The oval window was almost obliterated by otosclerosis. Only a small dimple in the mucous membrane remained, marking the location of the former round window.

The stapes head and remaining crura were removed. The oval window was re-opened with a diamond stone. A vein graft taken from the hand was placed over the oval window, and a piece of No. 90 polyethylene tubing was placed from the lenticular process of the incus to the vein graft after the method of Shea.

The round window was then opened with the diamond stone. It was found that a band of fibrous tissue embedded in bone led from the dimple on the promontory down to the former round window. At the end of the procedure it was possible to see movement of the fibrous tissue in the round window area when pressing on the incus.

Following surgery, the bone conduction remained the same, but he now had air conduction responses at 90 db for the speech frequencies. Using an air conduction aid his speech reception threshold was now 44 db.

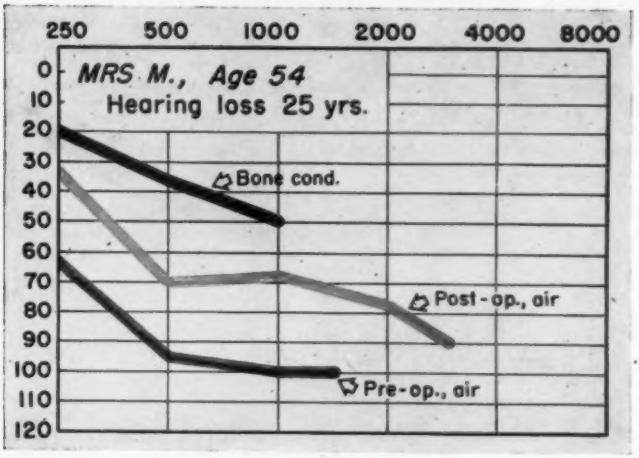
Following the above case, three additional cases with almost total deafness were done. These cases were also done under general anesthesia, but a trans-canal approach was used. This approach was found to give satisfactory visibility, and the surgery was shortened.

Two cases obtained a similar improvement to the above and one had no improvement.

Case 2. Mrs. M., a 54-year-old woman, first noted hearing loss at the age of 19. Fig. 4 shows the preoperative and postoperative audiograms.

In October, 1958, under local anesthesia, a trans-canal stapes mobilization approach was used. The round and oval windows were completely fixed by otosclerosis. The oval window was opened with a diamond stone after removal of the stapes. A vein graft and polyethylene strut were placed. At this point a hearing test was taken and no improvement noted. The round window was then opened with a diamond stone, and in this case it was necessary to open forward of the round window, since much of the scala tympani was involved in the otosclerosis. The window was considered open when perilymph escape was noted and when this showed pulsation with pressure on the incus. The patient immediately stated that the spoken voice was clearer, and her threshold improved.

Postoperatively the patient was nauseated and dizzy for several hours, but was able to leave the hospital the next day. Her threshold improved by 30 db as shown in Fig. 4, but the discrimination score dropped from 48 to 32. In addition, the patient could tolerate only 13 db of increased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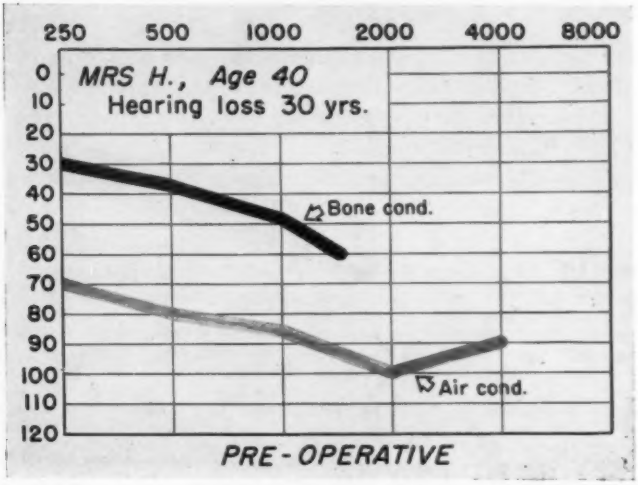
MRS M., Age 54
Hearing loss 25 yrs.
Bone cond.
Post-op., air
Pre-op., air

Fig. 4.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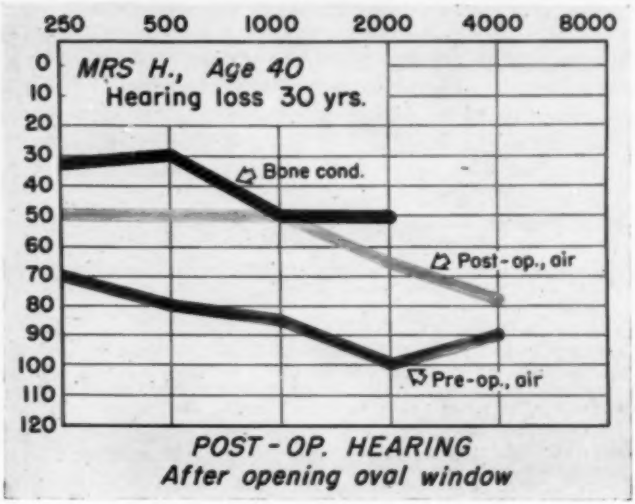
MRS H., Age 40
Hearing loss 30 yrs.
Bone cond.
Air cond.
PRE-OPERATIVE

Fig. 5-a.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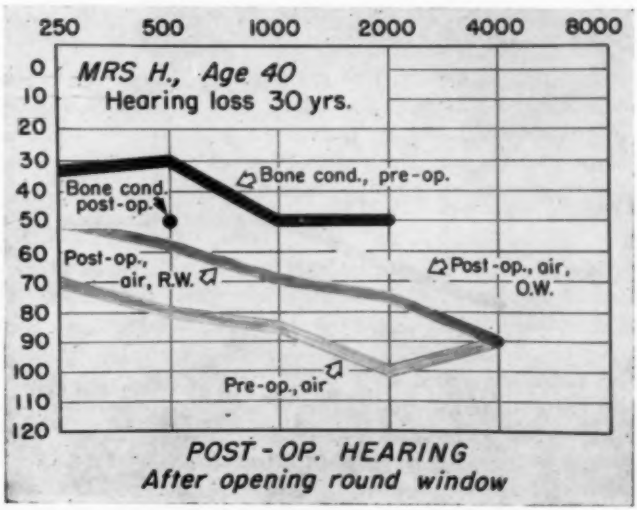
500
1000
2000
4000
8000
0
10
20
30
40
50
60
70
80
90
100
110
120
MRS H., Age 40
Hearing loss 30 yrs.
Bone cond.
Post-op., air
Pre-op., air
POST-OP. HEARING
After opening oval window

Fig. 5-b.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110
120
MRS H., Age 40
Hearing loss 30 yrs.
Bone cond., pre-op.
Bone cond. post-op.
Post-op., air, R.W.
Post-op., air, O.W.
Pre-op., air
POST-OP. HEARING
After opening round window

Fig. 6.

loudness. During the past months this has improved considerably, but the patient still prefers to wear her aid in the opposite ear.

In view of the results of the above case, it was decided to make a two-step operation out of the procedure. The first stage would be to open the oval window and the second stage to open the round window. It was felt that in this way less perilymph would be lost and less operative trauma would occur.

Case 3. Mrs. H., a 44-year-old woman, first noted hearing loss at the age of 14 years. Preoperative audiograms are shown in Fig. 5.

In November, 1958, the oval window was opened, using a diamond stone and a polyethylene strut. No vein graft was placed. This procedure resulted in a substantial gain in hearing.

One month later, using local anesthesia and a stapes approach, the round window was opened. This resulted in an immediate drop in hearing of 20 db, although the patient had no vertigo. The next morning her hearing was back to the preoperative level, so it was assumed that the immediate operative loss was due to loss of perilymph.

One month following surgery, however, the hearing level was down 10 db by air and the bone conduction decreased, as shown in Fig. 6. In addition, the patient had decreased loudness tolerance.

DISCUSSION.

At present the exact clinical effect of closure of the round window is not known. Wever and Laurence[6] were able to show air conduction losses as great as 20 db below 1,000 cps, from closure of the round window in experimental animals. They emphasize, however, that a displacement of only one-third of a cubic mm. is necessary to give normal hearing; therefore, clinically, we must have complete bony closure to have any effect.

Goodhill, et al.,[7] in animal experiments, was able to show a loss of approximately only 10 db by bone and air for the speech frequencies by closure of the round window. He is the only investigator to measure bone conduction in animals with closed round windows.

Work to date indicates that round window surgery in far advanced otosclerosis is a worthwhile procedure. The last two cases, however, indicate that in less advanced cases we must proceed with caution.

SUMMARY.

1. Regardless of the degree of otosclerotic fixation, the oval window can now be opened by means of a diamond drill technique.

2. The round window can also be opened surgically, but further investigation needs to be done to establish the indications and contraindications of this procedure.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES.

The 1959-1960 Home Study Courses in the Basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, executive secretary-treasurer of the Academy, 15 Second Street S. W., Rochester, Minn. Registration should be completed before August 15.

RADICAL NECK DISSECTION.

A Clinico-Pathological Study of 200 Cases.*†

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WERNER MUELLER, M.D.,
and
JAMES B. SNOW, JR., M.D.,
(By Invitation),
Boston, Mass.

In the evolution of otolaryngology, the radical neck dissection is a fairly recent development. As short a time ago as 1956, one of us (G.F.R.) reported 75 cases of radical neck dissection which had been done at the Massachusetts Eye and Ear Infirmary between the years 1948 and 1955.[7] Since that time there has been a marked acceleration in the utilization of this procedure in the treatment of cancer of the head and neck. Coincident with the frequent utilization of this operation, one of us (W.M.) in his role as director of otolaryngic pathology at the Massachusetts Eye and Ear Infirmary, recognized the need and importance of a careful dissection and study of the surgical specimens from these operations. Beginning in 1952, he initiated a time-consuming and painstaking study of each surgical specimen, in which each specimen on arrival at the laboratory, is carefully traced as to its outlines and landmarks, and a color photograph is made of each specimen. After fixation, each specimen is carefully and laboriously dissected to remove all recognizable lymph nodes. Indeed, this laboratory dissection of the specimen frequently takes as long or longer than the operation. As the laboratory dissection proceeds, each identifiable node is drawn on the previously traced outline to show its exact location and size. The nodes are classified anatomically as upper jugular, mid-

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jugular, lower jugular, sub-mental submaxillary, spinal accessory, and supraclavicular. Figs. 1 and 2 demonstrate the photograph of the original specimen and the laboratory drawing made on arrival at the laboratory. It will be seen that all dissected nodes are located in the drawings; and after the microscopic sections of each of these nodes are processed, positive nodes are marked accordingly to delineate them from non-malignant nodes. These efforts in conjunction with the clinical material available in the patients' records in the en-

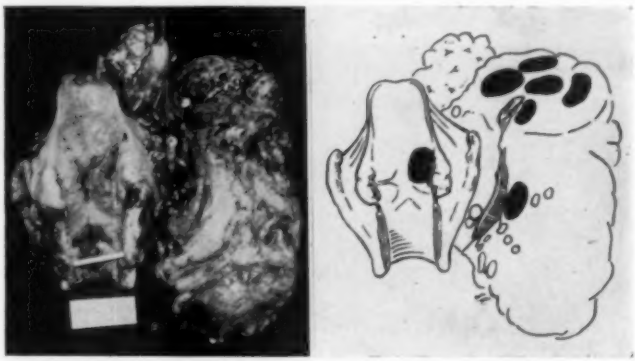

Fig. 1.

suing years have yielded a series of radical neck dissection cases on which we have an unusually extensive amount of information, both as to clinical course and surgical-pathological data. It is the purpose of this paper to present these findings.

Of necessity much of the information presented will be of statistical value only with no immediate clinical application; nevertheless, we feel that the material is of sufficient importance to be recorded in some detail for possible future use.

DESCRIPTION OF SAMPLE AND GENERAL OBSERVATIONS.

Our sample consists of 200 unselected patients who had radical neck dissection between 1948 and 1958. Twenty-three of the patients had bilateral dissections, and, therefore, the

pathological material consists of 223 neck specimens. Seventy of the neck dissections were performed as combined procedures and 153 as delayed operations with a time interval between treatment of the primary lesion and the subsequent radical neck dissection. There were 178 males and 22 females in the group. Table I demonstrates the age distribution and, as expected, shows a predominance in the 50-70 year age groups.

Table II depicts the distribution of the sample by site of the primary lesion. It will be noted that the predominance

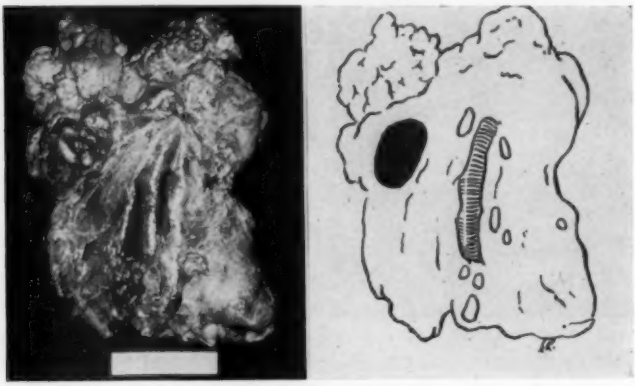

Fig. 2.

of cases in this series originated in the laryngeal and hypopharyngeal region. The classification of laryngeal and hypopharyngeal lesions for statistical purposes is still a matter of much debate; therefore, we are using an original classification based on the areas involved by the primary lesion. This classification with the number of cases in each area is shown diagramatically in Fig. 3. The areas illustrated are concerned, not with the region of supposed *origin* of the tumor, but with the areas *involved by* the tumor. It will be noted that for clarity and ease of understanding 14 minor areas of involvement have been condensed into four main groups of involvement, *i.e.*, cordal-subglottic, vestibular, supravestibular, and

TABLE I.

Sex:	
Male	178
Female	22
Age:	
30	0
31-40	5
41-50	28
51-60	72
61-70	69
71-80	24
81-90	2
Total	200

TABLE II.

Classification of Site of Primary Showing Number of Cases in Each Area and Group.

Area	Number of Cases		Area Involved	Group
A	9	21	Cordal	Cordal-Subglottic
B	1		Subglottic	
C	11		Cordal and Subglottic	
D	6	31	Supracordal Endolaryngeal	Vestibular
E	18		Cordal and Supracordal Endolaryngeal	
F	7		Cordal and Supracordal Endolaryngeal and Subglottic	
G	12	61	Epiglottic	Supra-vestibular
H	13		Base of Tongue	
I	19		Supracordal Endolaryngeal and Epiglottis	
J	10		Cordal and Supracordal Endolaryngeal and Epiglottic	
K	7		Epiglottic and Base of Tongue	
L	18	63	Hypopharyngeal	Hypopharyngeal
M	3		Hypopharyngeal and Base of Tongue	
N	42		Hypopharyngeal and Supracordal Endolaryngeal	
Other combinations	8			
Total laryngeal and hypopharyngeal	184			
	1		Nasopharynx	
	3		Eye	
	3		Nose	
	3		Ear	
	2		Parotid	
	1		Floor of mouth	
	3		Palate	
Total	200			

hypopharyngeal. The *cordal-subglottic group* includes lesions involving only the true cords, lesions involving only the subglottic area, and lesions involving both the true cords and subglottic area. The *vestibular group* includes lesions involving only the supracordal endolaryngeal area, lesions involving the true cords and the supracordal endolaryngeal area, lesions involving the true cords and the supracordal endolaryngeal area as well as the subglottic area. The *supravestibular group* includes lesions involving the epiglottis only, lesions involving the base of the tongue only, lesions involving the supracordal

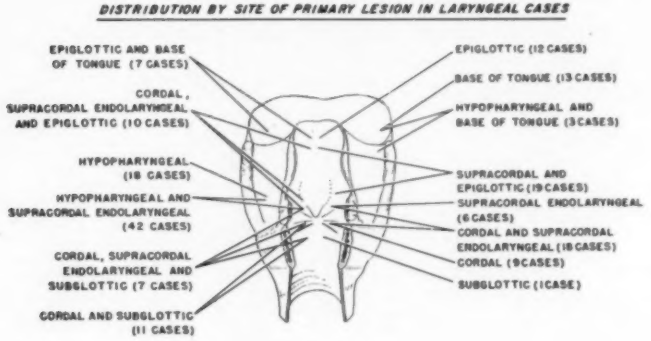

Fig. 3.

endolaryngeal area and the epiglottis, lesions involving the true cords as well as the supracordal endolaryngeal area and the epiglottis, and lesions involving both the epiglottis and the base of the tongue.

The *hypopharyngeal group* includes lesions involving only the hypopharynx, lesions involving the hypopharynx and the base of the tongue, and lesions involving the hypopharynx and the supracordal endolaryngeal area. These groups and the number of cases in each group are graphically shown in Fig. 4.

Wherever the site of the primary lesion is considered in our findings, we will present the abbreviated four region

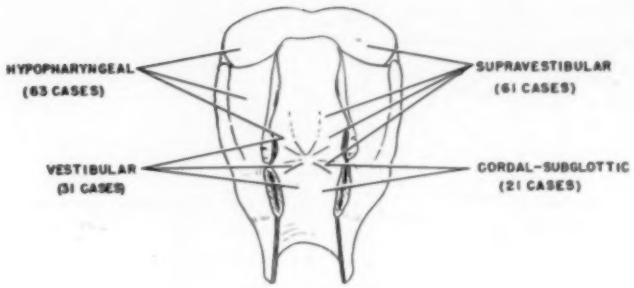

Fig. 4.

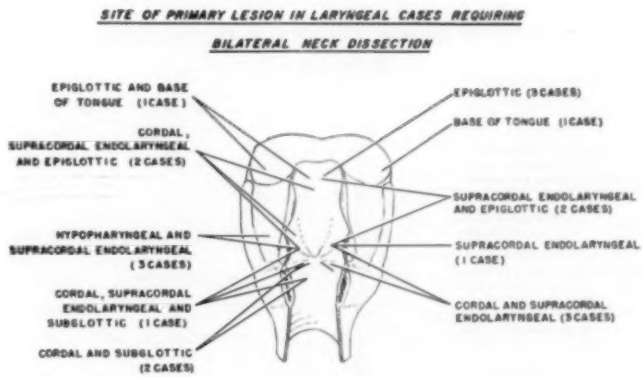

Fig. 5.

designations as well as the total laryngeal and hypopharyngeal classification to make our material as valuable as possible to future observers when perhaps a generally accepted classification for these lesions is adopted.

Fig. 5 shows the site of the primary lesion in 23 cases of bilateral radical neck dissection.

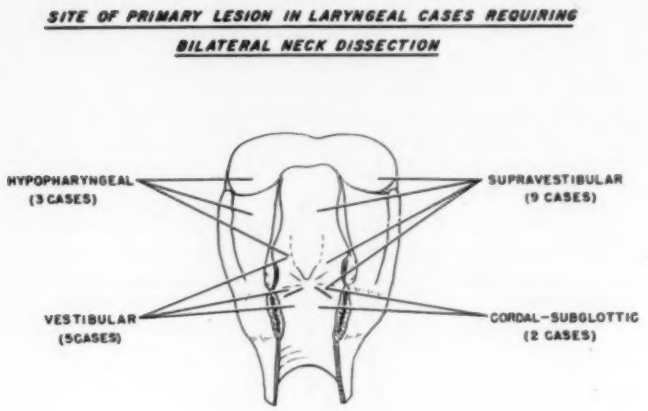

Fig. 6.

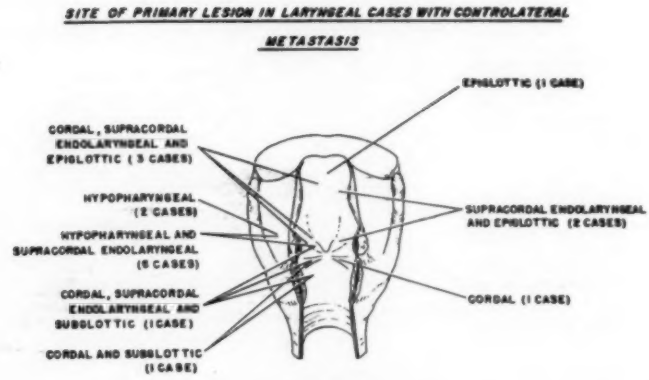

Fig. 7.

Fig. 6 demonstrates that the incidence of bilateral metastasis of cordal-subglottic lesions was 10 per cent; of vestibular lesions, 16 per cent; of supra-vestibular lesions 15 per cent, and of hypopharyngeal lesions 5 per cent. From these data we conclude that bilaterality of metastases does not appear to be influenced by the site of the primary lesion.

While considering bilateral metastasis, we also tabulated the number of cases of contralateral metastasis which did not first have an ipsilateral metastasis. We found that contralateral primary metastasis occurred in 17 cases or 9 per cent of the laryngeal and hypopharyngeal cases. Figs. 7 and 8 demonstrate the site of the primary lesion in these cases of contralateral metastases. It will be seen that 47 per cent, or nearly one-half of these cases involved the hypopharynx, and

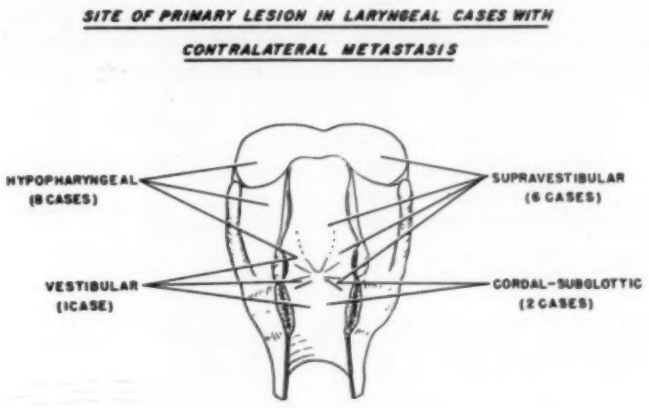

Fig. 8.

that 35 per cent involved the supravestibular area with involvement of the epiglottis in each case. Contralateral metastasis was rare from lesions within the lumen of the larynx.

While gathering information on the total sample, we thought it would be of interest to present our findings regarding extra laryngeal involvement in those patients on whom laryngectomy was performed and on whom we had careful studies of the laryngeal specimen. This group consisted of 153 carefully dissected larynges. The pre-cricoid or cricoid-thyroid node was identified in 39 cases and found positive in seven, or 18 per cent of these cases. It appears that in the process of laryngectomy it would be well to avoid dissection of this area of the larynx; and if the larynx is removed from below

upward, the operator should be careful not to allow a tenaculum to penetrate the region of the cricoid-thyroid node, else the metastatic tumor cells in this node might be disseminated into the field. We found that the pre-epiglottic space showed evidence of tumor extension in 24 cases, or 16 per cent. This fact would certainly bear out the necessity of removing the entire hyoid bone during laryngectomy, or at least that portion of the medial section of the hyoid bone forming the anterior limits of the pre-epiglottic space. Extension to involve the ala of the thyroid cartilage was found in 17 cases, or 11 per cent, and the strap muscles were found to be positive in eight cases, or 5 per cent. These data would certainly appear to substantiate the necessity for "wide field laryngectomy" with

TABLE III.

Pathological Diagnosis of Primary Lesion.

Squamous Cell Carcinoma, Grade I	27	60 Low Grade
Squamous Cell Carcinoma, Grade II	33	
Squamous Cell Carcinoma, Grade III	117	149 High Grade
Squamous Cell Carcinoma, Grade IV	19	
Undifferentiated Carcinoma	13	
Other	14	
Total	223	

removal of the strap muscles during routine laryngectomy. The thyroid gland showed evidence of involvement in 3 per cent of the cases, but was removed in only 27 cases. In these 27 cases, however, the gland was involved in 13 per cent which would seem to indicate that during the course of laryngectomy or of laryngectomy and radical neck dissection, if there is suspicion of the gland's being involved, it definitely should be removed *en bloc* with the larynx and neck specimen.

Table III demonstrates the pathological diagnosis in the total group of 223 neck dissections. From Fig. 9 it will be seen that there were over twice as many relatively high grade lesions as there were low grade lesions.

Table IV depicts the treatment of the primary lesion. This Table is presented for descriptive purposes only. We did not feel that it was the province of this presentation to enter

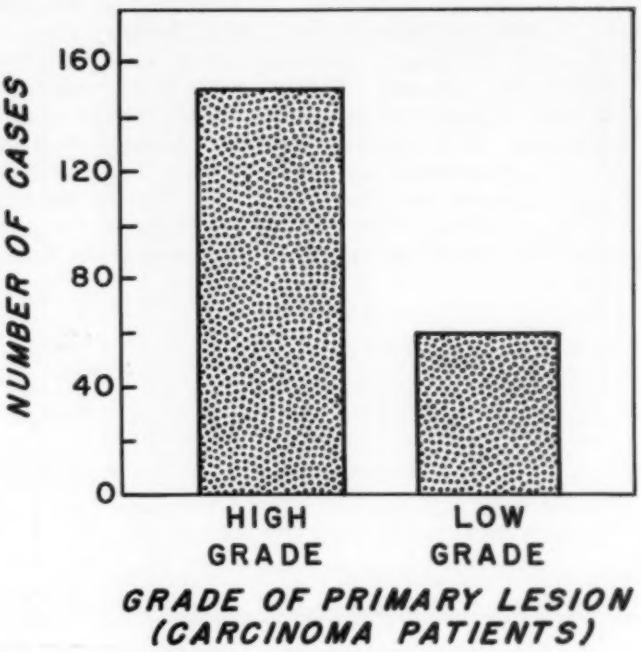

Fig. 9.

TABLE IV.

Type of Treatment of Primary Lesion.

Type of Treatment	Number
External Radiation	16
Radium	1
Hemiglossectomy	1
Hemiglossectomy and Laryngectomy	2
Combined Hemiglossectomy and Neck Dissection	5
Hemiglossectomy, Laryngectomy and Neck Dissection	2
"Commando"	2
Laryngo-fissure	3
Laryngo-fissure and Laryngectomy	2
Laryngectomy	72
Combined Laryngectomy and Neck Dissection	70
Laryngectomy and External Radiation	11
Other Surgical Excisions	12
None	1
Total	200

into a discussion or presentation of findings regarding the relative merit of radiation, surgery of the primary alone, or surgery of the primary in combination with neck dissection, or surgery plus radiation. Therapy would indeed constitute a formidable presentation by itself which we hope to compile in the future. It is interesting to note, however, that the number of laryngectomies alone and of combined laryngectomy and neck dissection are essentially equal. During the time that these operations were performed relatively few prophylactic neck dissections were being performed at the Massa-

TABLE V.

Time Between Onset of Symptoms and Treatment of Primary Lesion.

Time in Months	No. of Cases
0-1	10
1-2	27
2-3	27
3-4	21
4-5	22
5-6	18
6-7	8
7-8	4
8-9	6
9-10	0
10-11	2
11-12	16
Over a Year	30
No Information	9

chusetts Eye and Ear Infirmary; therefore, this data indicates the large number of cases in which palpable neck nodes are present at the time the need for laryngectomy is recognized.

Table V and Fig. 10 depict the time intervals between the onset of the patient's symptoms and the treatment of the primary lesion. Only 65 per cent of these patients were treated within six months of the onset of symptoms, and 23 per cent did not receive therapy for the primary lesion until 11 months or more after the onset of symptoms. The Table again demonstrates the disease's insidious nature, the patient's fear in seeking treatment, and perhaps on occasion the physician's lack of alertness in detecting early cancer.

Table VI and Fig. 11 are very interesting and clinically

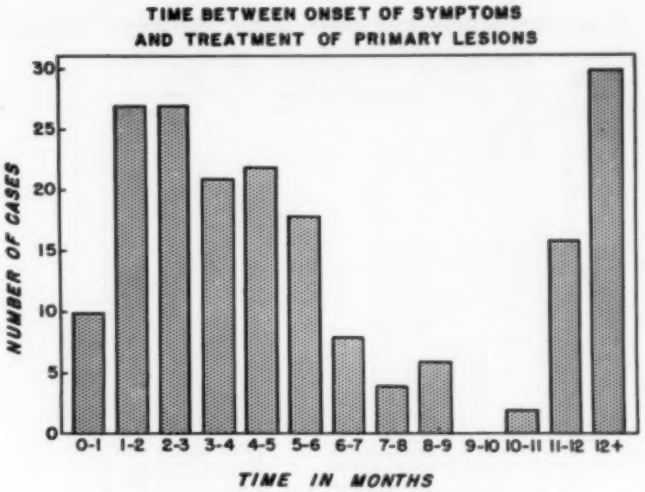

Fig. 10.

TABLE VI.

Time Between Treatment of Primary Lesion and Detection of Metastasis.

1-2 Months	29	52%	75%	89%
2-4 Months	16			
4-6 Months	22			
6-8 Months	10	23%		
8-10 Months	5			
10-12 Months	13			
1-2 Years	18	14%		
2 Years	15	11%		

applicable. They demonstrate the time elapsed between treatment of the primary lesion and detection of metastasis in those cases in which neck dissection was not done in combination with treatment of the primary lesion. It will be seen that the metastasis in over half of the cases was detected within six months of treatment of the primary, 75 per cent within one year, and almost 90 per cent within two years. Only 10 per cent metastasized after two years, and 4 per cent between two and five years.

Table VII compares the preoperative clinical estimate of

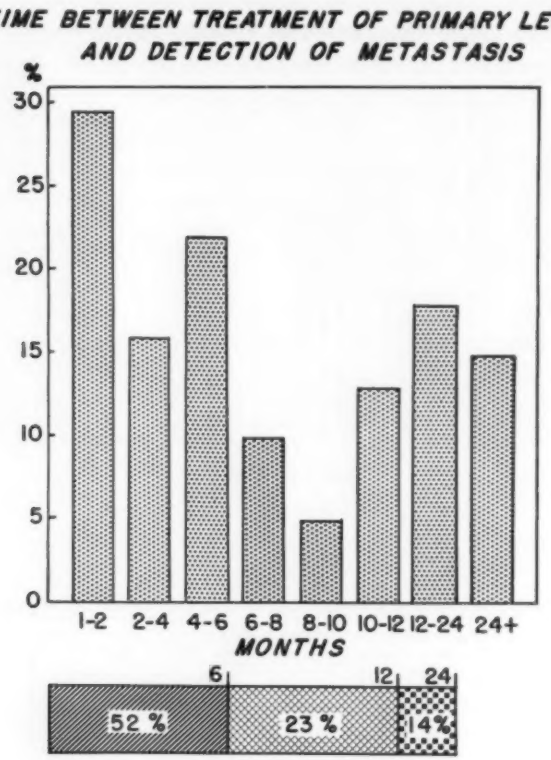

Fig. 11.

the metastasis with the actual size of the largest metastasis found in the laboratory dissection of the specimen. In 93 patients there was available a definite preoperative estimate of the metastasis. The preoperative estimate was correct in only 28 per cent of the cases; however, the estimate was within 1 cm. one way or the other in 72 per cent of the cases. When estimated incorrectly, the estimated size was smaller than the actual size in 35 per cent of the cases, and larger than the actual size in 65 per cent of the cases. More encouraging to the clinician performing a palpation of the neck,

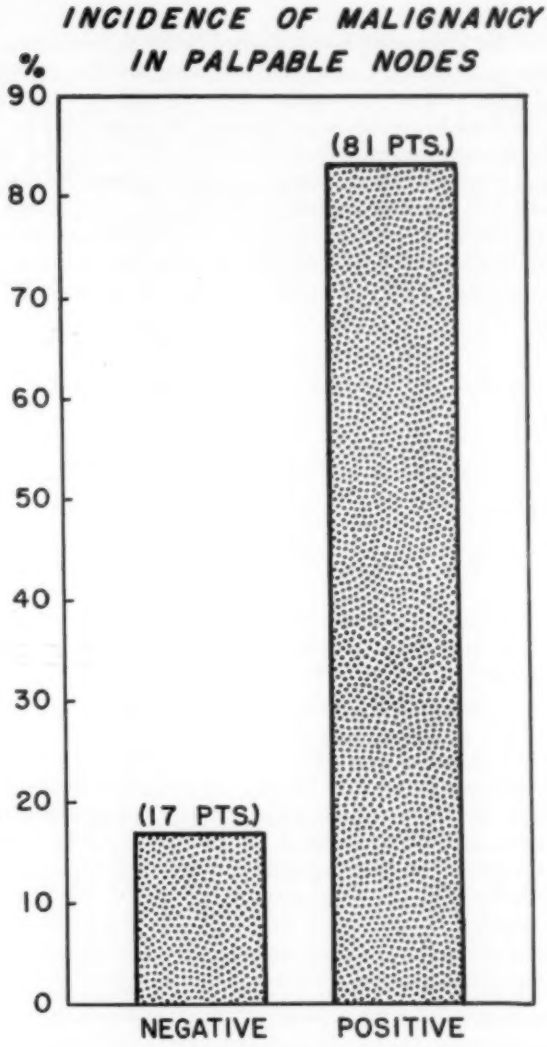
INCIDENCE OF MALIGNANCY
IN PALPABLE NODES
%
90
80
70
60
50
40
30
20
10
0
(81 PTS.)
(17 PTS.)
NEGATIVE
POSITIVE

Fig. 12.

however, was the finding regarding pathologic evidence of metastasis in clinically palpated nodes. As shown in Fig. 12 we found the preoperatively palpated nodes showed metastasis in 83 per cent of the cases, and the clinician was in error in only 17 per cent of the cases. The reliability of palpation of the neck as to presence or absence of metastasic tumor is a very important point regarding the whole matter of biopsying suspicious or questionable cervical metastasis in cases who have had a known primary malignancy in the head and neck. One of us (G.F.R.) in a previous publication[7] expressed vigorous opposition to the practice of biopsying cervical nodes in view of the great potential danger of dissemination of

TABLE VII.

Comparison of Estimate and Actual Size of Nodes.

Actual Size	Estimated Size				
	4 cm.	3 cm.	2 cm.	1 cm.	Total
4 cm.	8	8	10	4	30
3 cm.	5	2	9	5	21
2 cm.	3	5	6	7	21
1 cm. or less	1	5	4	11	21
Total	17	20	29	27	93

cancer cells and urged the clinician to have the courage of his convictions and to proceed with radical neck dissection rather than precede this procedure by biopsy. The above findings would indicate that if a node is palpable there is only a 17 per cent chance of its being negative in the presence of a previous or concomitant primary lesion, and this relatively small chance does not justify the potential dissemination and spread of tumor cells by biopsy. If in doubt, and a biopsy is to be considered, it should be in the form of a frozen section biopsy with all preparation made for a performance of a definitive radical neck dissection.

EFFECT OF THE DEGREE OF MALIGNANCY ON METASTASIS.

After making the above general observations, we turned our attention to the effect of the pathological diagnosis of the primary lesion upon the various factors concerning cervical metastasis.

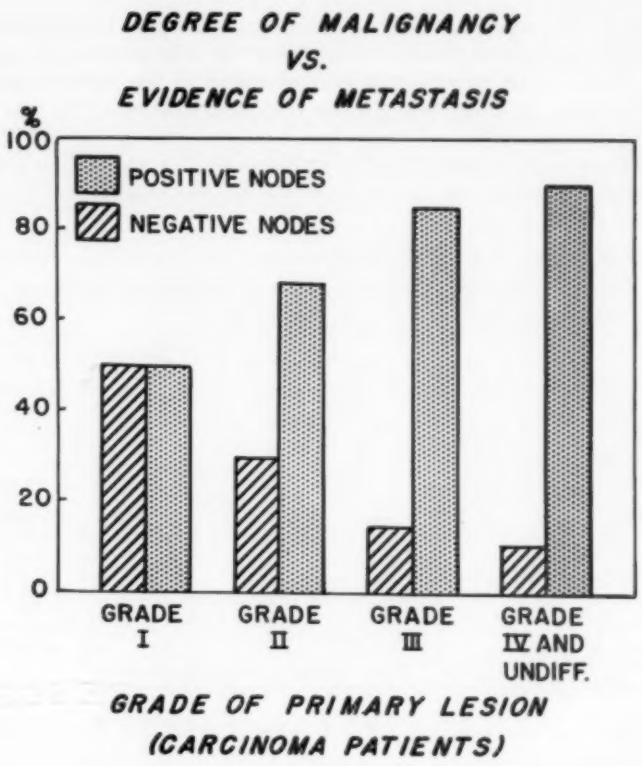

Fig. 13.

Correlation was made between the pathological diagnosis of the primary lesion and the location of the metastasis. This correlation was made in 99 cases in which the metastases were solitary or limited to one area. A study of these findings indicated that there was no apparent relationship between the pathological diagnosis or degree of malignancy of the primary lesion and the location of the metastasis.

We next studied the effect of the pathological diagnosis of the primary lesion upon the presence of metastasis and the maximum size of the metastatic nodes. It will be seen from

Table VIII and Fig. 13 that in patients in whom the primary lesion was a Grade I carcinoma, there was a 50 per cent chance of there being no evidence of metastasis in the radical neck specimen. In those with Grade II primary lesion there was a 33 per cent chance of there being no evidence of metastasis. In those with a Grade II primary lesion there was a 33 per cent chance of there being no evidence of metastasis. In those

TABLE VIII.

Correlation of Degree of Malignancy with Evidence of Metastasis.

Pathological Diagnosis of Primary	Maximum Size of Node				
	4 cm.	3 cm.	2 cm. or Less	No Evidence of Metastasis	Total
Grade I	5	3	6	14	28
Grade II	4	4	13	10	30
Grade III	24	32	30	15	101
Grade IV	7	5	3	2	17
Undifferentiated	5	2	2	1	10
Other	7	4	2	1	14
Total	52	50	55	43	200

TABLE IX.

Comparison of the Degree of Malignancy with the Number of Positive Nodes.

Grade of Primary Carcinoma	Number of Positive Nodes									
	Solitary	2	3	4	5	6	7	8	9	Total
I and II	20	5	1	0	4	3	1	0	1	36
III	58	13	10	7	3	3	1	0	5	100
IV and Undifferentiated	15	2	4	1	0	1	0	2	3	28

showing a primary lesion of Grade III or more, there was only an 11-15 per cent chance of there being no evidence of metastasis. Thus, it would appear that the greater the degree of malignancy of the primary lesion the more likely it is that dissectible cervical nodes will be positively involved. We could show no apparent relationship between the degree of malignancy of the primary lesion and the maximum size of the largest metastastic node.

We next studied the effect of the degree of malignancy of the primary lesion upon the total number of metastic nodes

or multiplicity of metastases. Table IX indicates that the degree of malignancy has no significant effect on multiplicity of metastases.

It appeared to us that another important relationship to be studied would be that between the degree of malignancy of the primary lesion and the time between the treatment of the primary lesion and detection of metastasis. Such a relationship might help us in determining the amount of diligence and frequency required in the postoperative observation following treatment of a primary lesion. Much to our surprise, as

TABLE X.

Effect of Degrees of Malignancy on Time of Subsequent Metastasis in Delayed Cases.

Time Between Treatment of Primary and Detection of Metastasis	Pathological Diagnosis of Primary						
	Grade I	Grade II	Grade III	Grade IV	Undifferentiated	Other	Total
<1-2 Months	4	1	13	4	3	4	29
2-4 Months	4	1	8	2		1	16
4-6 Months		5	15	2			22
6-8 Months		4	5		1		10
8-10 Months		1	2		1	1	5
10-12 Months	2	2	6	2		1	13
1-2 Years	2	1	10	2	2	1	18
>2 Years		5	7	2		1	15
Total	12	20	66	14	7	9	128

shown in Table X, there appeared to be no relation between the pathological diagnosis of the primary lesion and the time between the treatment of the primary and detection of the metastasis. These findings, of course, do not prove by any means that there is no relation between these two factors, because so many other factors were concerned in these patients such as the follow-up interval, the obesity of the patient, etc.; nevertheless, we could not demonstrate a definite relationship between these two factors.

EFFECT OF SITE OF THE PRIMARY LESION.

In order to determine the possible effects and influences of the site of the primary lesion, various correlations were

TABLE XI.

Comparison of Site of Primary Lesion with Presence and Multiplicity of Metastases.

Site of Primary	No Evidence Metastasis	Solitary Metastasis	Multiple Metastasis	Total		No Evidence Metastasis	Solitary Metastasis	Multiple Metastasis
Cordal-Subglottic Area								
Group A	4	5	0	9				
Group B	0	1	0	1	24	6	13	5
Group C	2	7	5	14				
Vestibular Area								
Group D	1	2	4	7				
Group E	4	9	8	21	35	8	12	15
Group F	3	1	3	7				
Supravestibular Area								
Group G	2	10	3	15				
Group H	7	5	2	14				
Group I	3	11	7	21	69	13	39	17
Group J	1	8	2	12				
Group K	0	5	2	7				
Hypopharyngeal Area								
Group L	4	6	8	18				
Group M	0	1	2	3	67	14	27	26
Group N	10	20	16	46				
Other Laryngeal	3	3	4	10				
Non-Laryngeal	4	5	9	18				
Total	48	99	76	223				

TABLE XII.
Effect of Primary Site on Maximum Size of Nodes.

Site of Primary	Maximum Size of Nodes									
	4 cm.	3 cm.	2 cm. or less	No Evidence Malignancy	Total		4 cm.	3 cm.	2 cm. or less	No Evidence Malignancy
Cordal-Subglottic Area										
Group A	2		3	4	9					
Group B			1		1	21	3	5	8	5
Group C	1	5	4	1	11					
Vestibular Area										
Group D	4	2			6					
Group E	8	3	3	4	18	31	14	7	3	7
Group F	2	2		3	7					
Supravestibular Area										
Group G	3	6	2	1	12					
Group H	1	1	4	7	13					
Group I	6	6	4	3	19	61	16	18	15	12
Group J	4	3	2	1	10					
Group K	2	2	3		7					
Hypopharyngeal Area										
Group L	4	3	7	4	18					
Group M	1	1	1		3	63	13	16	21	13
Group N	8	12	13	9	42					
Other Laryngeal	2		3	3	8					
Non-Laryngeal	4	4	5	3	16					
Total	52	50	55	43	200					

TABLE XIII.

Number and Location of Nodes by Site of Primary Lesion.

Site of Primary	Location and Number of Metastatic Nodes																			
	Upper Jugular	Middle Jugular	Lower Jugular	Submaxillary	Supraclavicular	Submental	Accessory	No Evidence Malignancy	Total Nodes		Upper Jugular	Middle Jugular	Lower Jugular	Submaxillary	Supraclavicular	Submental	Accessory	No Evidence Malignancy	Total Nodes	Total Cases	Nodes per Case
Cordal-Subglottic Area																					
Group A	2	1	1		2			4	6												
Group B					1				1	29	16	6	3	1	3			6	29	= 1.4
Group C	14	5	2	1				2	22										21	
Vestibular Area																					
Group D	8	3	4	3				1	18												
Group E	24	7	12	8			3	4	54	91	38	14	22	11			6	8	91	= 2.9
Group F	6	4	6				3	3	19										31	
Supravestibular Area																					
Group G	14	3	7	9		1		2	34												
Group H	5	3	3	2				7	13												
Group I	23	11	16				2	3	52	139	59	22	32	23		1	2	13	139	= 2.3
Group J	8	3	5	3				1	19										61	
Group K	9	2	1	9					21												
Hypopharyngeal Area																					
Group L	17	9	11		2		1	4	40												
Group M	6	1	1						8	138	67	34	20	5	11		1	14	138	= 2.2
Group N	44	24	8	5	9			10	90										63	
Other	9	3	3	1				7	16		9	3	3	1				7			
Total	189	79	80	41	14	1	9	48	413												

made. Comparison of the site of the primary lesion with the presence of metastasis in Table XI showed that the site of the primary lesion appeared to have no effect on whether palpable nodes contained malignant cells.

We also studied the relationship between the primary site and the maximum size of positive nodes and could demonstrate no relationship between these two factors. These findings are shown in Table XII.

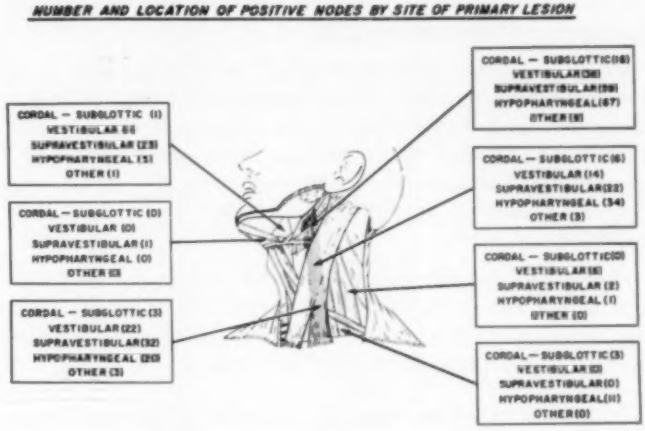

Fig. 14.

Comparison of the effect of the primary site upon the number and location of nodes is presented in Table XIII and Fig. 14. Table XIII is rather complex: it demonstrates the location of positive nodes and gives the sites of the primary lesion in detail so that the reader may make his own compilations and conclusions. In general, cordal-subglottic lesions appeared to have fewer nodes per case than did those of other sites, and vestibular lesions had the most nodes per case, ranking ahead of supravestibular and hypopharyngeal lesions in that order. Although the distribution of nodes to the upper jugular and middle jugular chains was fairly uniform throughout the various primary sites, there appeared to be a higher

incidence of involvement of the middle jugular nodes in hypopharyngeal lesions. Extension to involve the lower jugular nodes was fairly evenly distributed with somewhat less tendency of the cordal-subglottic lesions to metastasize to this area. It is interesting to note that the supravestibular lesions accounted for a much higher percentage of the cases showing submaxillary involvement than the other groups, while the hypopharyngeal lesions were responsible for most of the metastases to the supraclavicular nodes. Vestibular lesions accounted for two-thirds of the nodes involving the region of the spinal accessory nerve. In only one case was metastasis found in the submental region.

Occasionally the question is raised of the feasibility of doing somewhat less than what is now considered a total radical neck dissection, and Table XIII would indicate that there is sufficient involvement of the various areas encompassed by the presently accepted neck dissection in these 200 cases to make a total dissection mandatory whenever neck dissection is considered at all.

Table XIV reveals that there is no correlation between the site and grade of the primary lesion; therefore, it appears that the site of the primary influences the number of positive nodes per case independently of grading.

INFLUENCE OF THE SIZE AND LOCATION OF PRIMARY NODE ON SUBSEQUENT NODES.

We next turned our attention to a study of the influence of the primary metastatic node on subsequent or satellite nodes. In this regard we considered the largest node in the specimen to be the primary node, and in almost all cases we found that there was one node or group of nodes which constituted the main metastatic mass with scattered smaller or satellite nodes. We noted with interest that, of 99 cases where the metastasis clinically appeared as a solitary mass in 31 cases or 31 per cent, it was found on dissection to be a group of nodes in one place rather than a single node. A study of the influence of the size of the primary node on the total number of positive nodes is presented in Table XV. It will be seen that nodes 4 cm. or larger were found in 76 neck specimens, and in 47

TABLE XIV.

Comparison of Pathological Diagnosis of Primary and Site of Primary Lesion.

	Pathological Diagnosis of Primary Lesion													
	Grade							Grade						
Site of Primary	I	II	III	IV	Undifferentiated	Other	Total		1	II	III	IV	Undifferentiated	Other
Cordal-Subglottic Area														
Group A	3		3	2	1		9							
Group B			1				1	21	4	2	12	2	1	
Group C	1	2	8				11							
Vestibular Area														
Group D		1	3	1	1		6							
Group E	4	3	8	1		2	18	31	4	7	14	3	1	2
Group F		3	3	1			7							
Supravestibular Area														
Group G	1	2	8			1	12							
Group H	3		6	1	2	1	13							
Group I	2	2	11	3		1	19	61	6	6	37	5	3	4
Group J			10				10							
Group K		2	2	1	1	1	7							
Hypopharyngeal Area														
Group L	3	2	8	1	2	2	18							
Group M	1		2				3	63	10	10	32	4	2	5
Group N	6	8	22	3		3	42							
Other Laryngeal	2	1	4	1			8							
Non-Laryngeal	2	4	2	2	3	3	16							
Total	28	30	101	17	10	14	200		24	25	95	14	7	11

per cent of these there was a solitary metastasis, whereas in 53 per cent there were multiple metastases. Three cm. nodes were found in 40 necks, and in 52.5 per cent of these there was a solitary metastasis, and in 47.5 per cent there were multiple metastases. Nodes measuring 2 cm. or less were found in 59 specimens; and in 71 per cent of these there was a solitary metastasis, and in only 29 per cent were there multiple metastases. We feel from these findings and Table

TABLE XV.

Influence of Size of Primary Node on Total Number of Positive Nodes.

Size of Primary Node	Number of Nodes								
	Solitary Metastasis	2	3	4	5	6	7	8	9 or over	Total
4 cm.	36	4	7	6	7	7	1	2	6	76
3 cm.	31	6	4	3	2	1			3	40
2 cm. or less	42	9	2	1	2	2			1	59
Total	99	19	13	10	11	10	1	2	10	175

TABLE XVI.

Correlation of Size of Primary Node and Number of Metastatic Areas Involved.

Size of Primary Node	Number of Areas Involved in Metastasis				
	1	2	3	4	Total
4 cm.	36	23	12	5	76
3 cm.	21	11	7	1	40
2 cm. or smaller	42	13	4		59
Total	99	47	23	6	175

XV that the number of positive nodes and the tendency to multiple metastases increases in proportion to the size of the primary metastatic node.

We next studied the influence of the size of the primary node on the total number of areas involved in metastasis and these findings are presented in Table XVI. These findings indicate that the number of areas involved is in proportion to the size of the primary node. This relationship is in accord with the previous finding of a correlation between multiple metastases and size of the primary node.

We then made a tabulation of the influence of the site of

the primary node upon the location of subsequent nodes. This material is presented in Fig. 15. It is interesting to note that, if the primary node is in the upper jugular region, the secondary node will be in the middle jugular region 53 per cent of the time and in the lower jugular area 33 per cent of the time. This finding conforms with the usual thinking regarding spread of subsequent metastasis. We found, however, that

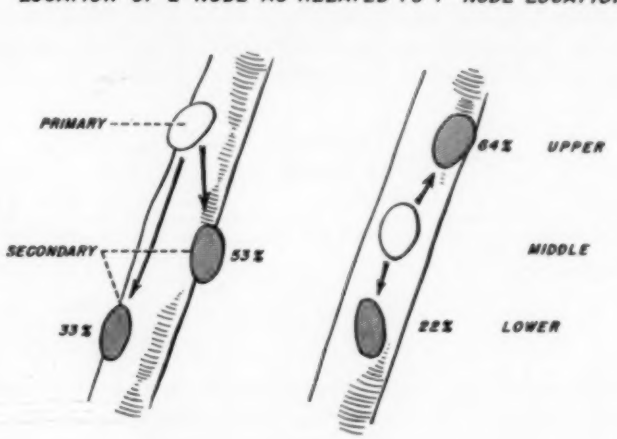

Fig. 15.

if the primary node is in the middle jugular area, the secondary node will occur in the upper jugular region 64 per cent of the time and in the lower jugular region only 22 per cent of the time.

SURVIVAL DATA.

Unfortunately, in this study we found that we did not have sufficient patients of potential four and five year survival to be of significance. Although we had numerous potential two year survivors, we did not feel that two years was a sufficiently long follow-up period and decided to base our survival statistics on 84 patients whose neck dissection had been per-

formed three or more years prior to this study, and thus, were potential three year survivors.

Table XVII demonstrates the overall survival figures. There are 21 cases (or 25 per cent) living with no evidence of recurrence three years or more after their radical neck dissection; however, 13 of these 21 had shown no evidence of malignancy on dissection of their neck specimens. Only eight, or 17 per cent of those patients whose neck specimens showed positive nodes have survived three years. Of the 23 patients on whom bilateral neck dissections were performed, there were ten potential three year survivors. Four of these patients were living without evidence of recurrence three years

TABLE XVII.

Overall Survival Data.

Living—No Evidence of Recurrence	21	25%
Living with Recurrence	5	6%
Dead of Disease	46	55%
Dead of Other Causes	6	7%
Lost to Follow-up	6	7%
Total	84	

after operation, five were dead of disease, and one had died of other causes. Five were living with no evidence of recurrence more than a year after surgery, and three more than two years after surgery. Three patients had died of disease less than a year following operation, and one died of disease less than two years following surgery. One patient of these 23 was lost to follow-up. There were four patients with bilateral neck dissections on whom there was no evidence of metastases on one side upon pathologic examination. Two of these patients are living without recurrence four years after surgery, and one two years after surgery. One was living with recurrence three years after surgery.

Following a fairly systematic plan of comparing survival figures against the data available from this study, we compared the three year follow-up data according to the site of the primary lesion. The results of these compilations are presented in Table XVIII and Fig. 16 which present a number

TABLE XVIII.

Survival According to Site of Primary Lesion.

Site of Primary Lesion	Living; No Evidence Recurrence	Living with Recurrence	Dead of Disease	Dead; Other Causes	Lost to Follow-up	Total		Living; No Evidence Recurrence	Living with Recurrence	Dead of Disease	Dead; Other Causes	Lost to Follow-up
Cordal and Subglottic Area												
Group A	2		2	1		5						
Group B					1	1	10	4 (40%)		4 (40%)	1	1
Group C	2		2			4						
Vestibular Area												
Group D		2	1			3						
Group E	6	1	1		2	10	18	7 (39%)	4	5 (28%)		
Group F	1	1	3			5						
Supravestibular Area												
Group G	1		4			5						
Group H	1	1	2	1	2	7						
Group I	1		4			5	20	3 (15%)	1	13 (65%)	1	2
Group J			1			1						
Group K			2			2						
Hypopharyngeal Area												
Group L			1	1		2						
Group M	1		2			3	22	5 (23%)		13 (59%)	3	1
Group N	4		10	2	1	17						
Other Laryngeal	1		4			5						
Non-Laryngeal	1		7	1		9						
Totals	21	5	46	6	6	84						

of interesting findings, the most important of which is that the percentage of patients living without recurrence is much higher in the cordal-subglottic and vestibular groups than in supravestibular and hypopharyngeal groups. In evaluating the patients who had died of disease within three years, it is seen that the supravestibular lesions account for the greatest percentage of these patients, and would, therefore, appear to carry the worst prognosis.

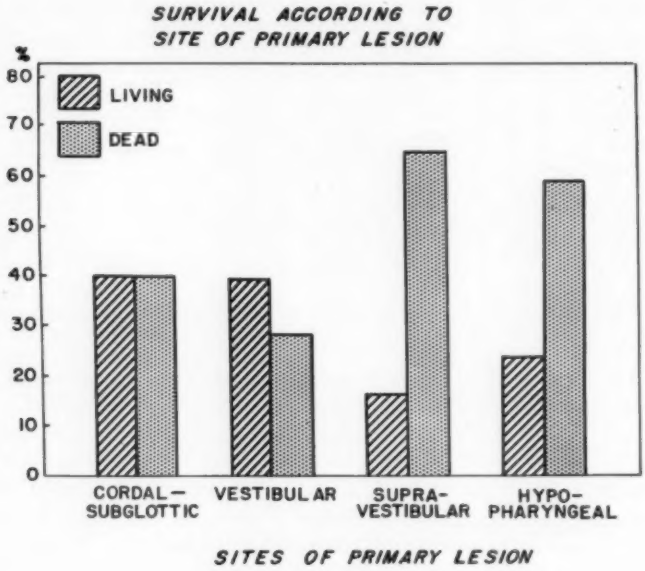

Fig. 16.

We were pleasantly surprised to find that five of 22 patients in the hypopharyngeal group were living with no evidence of recurrence three years following surgery, and that four of these five constituted large lesions of the pyriform sinus with extension into the endolarynx. It is fairly well accepted that lesions which are limited to the endolarynx have a much better prognosis than supravestibular and hypopharyngeal lesions.

It is very encouraging, however, to find hypopharyngeal lesions showing a 25 per cent three year survival rate with the use of neck dissection.

A study of the effect of location of the primary node upon survival revealed that the location of the primary node does not appear to affect survival.

Table XIX demonstrates the effect of the actual size of the primary node upon survival. A study of this Table reveals that there appears to be no correlation between the actual size of the primary node and survival. This finding was especially surprising and rather dismaying, since one of us (G.F.R.)

TABLE XIX.

Survival According to Actual Size of Primary Node.

Site of Primary Node	Living; No Evidence of Recurrence	Living with Recurrence	Dead of Disease	Dead of Other Causes	Lost to Follow-up	Total
4 cm.	5	4	14	1	2	25
3 cm.	3		10	1		14
2 cm. or less	3		16	1	4	24
No Evidence of Metastasis	10	1	6	4		21
Total	21	5	46	6	6	84

had previously shown a definite relationship between the size of the primary node and survival in a study based on 75 patients using estimated size of nodes and two year survival.[7] In an attempt to reconcile the variation between the findings in Table XIX and our previous findings, we compared the actual size of the node and the estimated size to the two year, three year, four year and five year survival findings in Table XX. (We hasten to point out that numbers in Table XX differ from those in Table XIX because Table XX is computed in years while the concise three year survival rates in all other tables were computed in months).

Although a study of Table XX appears to indicate a slight tendency toward a better prognosis when the estimated size of the node is less than 2 cm., it is certainly not the striking difference we found in our previous study. We still feel that such a relationship does exist, since we demonstrate elsewhere

TABLE XX.

Survival by Actual and Estimated Size of Node.

Primary Node	2 Years or Less		3 Years		4 Years		5 Years		Total
	Living; No Recurrence	Dead	Living; No Recurrence	Dead	Living; No Recurrence	Dead	Living; No Recurrence	Dead	
Actual Size—									
4 cm.	15	16	6	2		0	1	1	41
3 cm.	17	18	2	1	3	0			41
2 cm. or less	14	19	5			0	1	1	40
Total	46	53	13	3	3		2	2	122
Estimated Size—									
4 cm.	6	7						1	20
3 cm.	9	7		2	1			1	20
2 cm. or less	23	19	5		3		2		52
Total	38	33	5	2	4		2	1	85

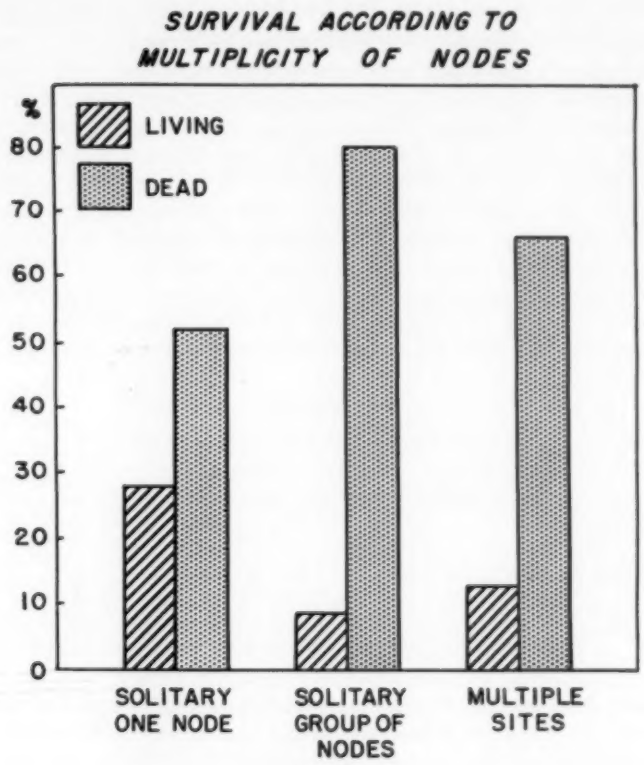

Fig. 17.

in this paper a direct relationship of node size on the tendency to multiplicity of metastases, total number of nodes and to the number of areas of cervical involvement, each of which in turn appear to affect survival; therefore, we have indirect evidence that survival decreases with node size although we cannot demonstrate it directly as we did in our previous study. Clinically, it would certainly appear that the prognosis is much more grave in large metastatic nodes than in smaller ones, and to help resolve this problem we are presently engaged in reappraisal of the original 75 patients.

We next studied the survival of these patients with reference to whether the metastasis was a solitary node, a group of nodes in one place, or nodes in more than one area. This tabulation shown in Table XXI and Fig. 17 reveals some very interesting findings. The predominance of patients living with no evidence of recurrence for three years is in the cases in which the metastasis was limited to one node. The survival rate of cases with only one node (28 per cent) is more than twice that of cases with more than one node in a single location (7 per cent). This finding is confirmed by the fact that multiple metastases constitute more than twice as many cases dead of disease as does solitary involvement. This tendency to a poor prognosis where there are multiple metas-

TABLE XXI.

Survival According to Multiplicity of Nodes.

	Living; No Evidence of Recurrence	Living with Recurrence	Dead of Disease	Dead of Other Causes	Lost to Follow-up	Total
Solitary— One Node	7	1	13	1	3	25
Solitary— Group of Nodes	1	0	12	1	1	15
Multiple Metastases	3	3	15	0	2	23
No Evidence of Malignancy	10	1	6	4	0	21

tases is in agreement with the findings of Taylor and Nathanson.[8] It is also in agreement with a "pet theory" of ours that metastatic cancer is contained within the first or primary metastatic node for a fairly long period of time and is more definitely curable until the node reaches a certain size. At this time the cancer breaks through the capsule of its node to form a suddenly larger metastatic mass or to produce adjacent satellite nodes. Once these changes occur the prognosis drops precipitously. These phenomena might also account for the sudden appearance of rather large nodes which we have all seen clinically.

We next wondered, in view of present day difficulties in obtaining hospital beds and operating time, what effect the time between detection of metastasis and definitive surgery would have upon survival of these patients. Table XXII in-

TABLE XXII.

Survival Based on Time Between Detection of Metastasis and Surgery.

Time Between Detection of Metastasis and Surgery	Living: No Evidence of Recurrence	Dead of Disease	Other	Total
1 Week	12 (25%)	23 (48%)	13	48
2 Weeks	5 (30%)	8 (61%)		13
3 Weeks		3 (70%)	1	4
4 Weeks	2 (22%)	5 (55%)	2	9
Other			10	10
Total	19	39		84

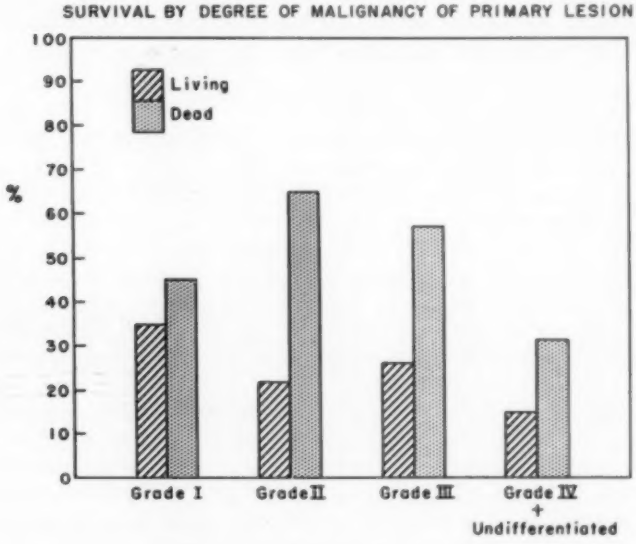

Fig. 18.

dicates that it makes no apparent difference in the three year survival rate whether the operation is performed one week or as long as four weeks after detection of metastasis.

Although the grade of the primary lesion had shown no appreciable effect on metastasis, we wondered whether it would affect survival. The results of this correlation are shown in Table XXIII and Fig. 18. Although there appears

to be a higher percentage of patients living without recurrence in low grade lesions, this relationship is not confirmed in the group dead of disease.

DISCUSSION.

In the course of this investigation we have come up with a number of positive and negative findings with emphasis on the latter. These findings have raised almost as many questions as they have answered. We will discuss the more important of our findings and present our conclusions and opinions regarding the answers they have yielded and the questions they have raised.

TABLE XXIII.

Effect of the Degree of Malignancy of the Primary Lesion on Survival.

	Living; No Evidence of Recurrence	Living with Recurrence	Dead of Disease	Dead of Other Causes	Lost to Follow-up	Total
Grade I	7	1	9	0	3	20
Grade II	5	0	15	2	1	23
Grade III	7	1	15	2	1	26
Grade IV and Undifferentiated	2	3	5	2	1	13
Other	0	0	2	0	0	2
Total	21	5	46	6	6	84

Regarding contralateral metastasis, we found that nearly half of the cases involved the hypopharynx, and a third of the cases were of supravestibular origin, involving the epiglottis. Contralateral metastasis was rare from lesions within the lumen of the larynx. Although based on a relatively small number of cases, these findings might be of value clinically to those who favor prophylactic neck dissection on the contralateral side. It would appear from our figures that prophylactic contralateral dissection might be indicated in cases of hypopharyngeal or supravestibular primaries, but would be less justified in lesions arising in the lumen of the endolarynx.

The cricothyroid lymph node was positive in 18 per cent of the 39 cases in which it was identified, and accordingly it would appear wise to avoid instrumentation in the region of

this node at the time of laryngectomy. The ala of the thyroid cartilage was invaded in 17 cases, or 11 per cent. Since tumor involved the preepiglottic space in 16 per cent and the strap muscles in 5 per cent of our laryngectomy specimens, it would seem mandatory that a laryngectomy should be of the "wide field" type, including resection of the greater portion of the hyoid bone and the strap muscles to insure removal of the tumor in these sites.

Our findings regarding the thyoid gland are of insufficient number to draw conclusions. In this series the thyroid gland was removed only when there was suspicion at time of operation that it was involved. The specimens showed positive involvement in 13 per cent of those cases in which it was removed. Ogura found involvement of the thyroid gland in 10 per cent of his cases.[4] Although we have insufficient data on which to draw conclusions, our opinion is that in the course of combined laryngectomy and radical neck dissection it is probably better cancer surgery to excise the thyroid lobe. Conversely, in the performance of a total laryngectomy in the absence of suspicion of thyroid gland involvement it should not be excised. One lobe can then be removed at the time of subsequent radical neck dissection without difficulties of hypoparathyroidism should metastasis occur.

In our laryngectomized patients there was essentially an equal number of combined laryngectomy-neck dissections and laryngectomies alone. Since very few prophylactic neck dissections were done during the period of this study, it would appear that there is a high incidence of metastasis at the time primary laryngeal lesions are discovered. Only 65 per cent of the patients in this series were treated for their primary lesion within six months of the onset of symptoms, and 23 per cent received therapy eleven months or more after the onset of symptoms.

Over one-half of these patients demonstrated cervical metastasis within six months of the treatment of their primary, 75 per cent within a year, and almost 90 per cent within two years. In only 10 per cent did metastasis occur after two years. We feel that these are very comforting statistics to

quote patients who have passed their one and two year post-operative periods.

Another interesting finding was the unreliability of the preoperative estimate of node size. We found that the preoperative estimate was correct in only 28 per cent of our cases in which this information was available. It is somewhat comforting to the surgeon to know, however, that, when wrong, the estimate was within 1 cm., one way or the other, in 72 per cent of the cases. It is also comforting to know that when estimated incorrectly, the estimated size was smaller than actual size in only a third of the cases and larger than actual size in two-thirds of the cases. In essence, when estimating node size, the surgeon usually estimates the node larger than it actually is, but he is usually within 1 cm. of its actual size.

As Taylor and Nathanson[8] have pointed out, "Enlargement or palpability of nodes is the most dependable guide to the presence of metastasis, and the other characteristics of nodes are secondary to it." We were pleased to be able to confirm this point by our finding that palpable cervical nodes in patients with known head and neck primary cancer, show evidence of metastasis 83 per cent of the time. In other words, the clinician will be in error only 17 per cent of the time. This finding is also quite consistent with the overall error of 16.4 per cent in Kuhn's study.[2] As stated earlier, this finding strengthens our previous feeling that biopsy of cervical nodes in patients with known head and neck primary cancer is to be condemned. If there are serious doubts regarding the palpable node, a frozen section biopsy should be done with preparation for a definitive radical neck dissection; however, it is preferable to have the courage of one's convictions and proceed with neck dissection, and our findings indicate that it will be proper therapy 83 per cent of the time.

Effect of Degree of Malignancy.

In this group of 200 patients there were twice as many lesions of relatively high grade malignancy as of low grade; moreover, it appears that the greater the degree of malignancy of the primary lesion, the more likely it is that dissectible nodes will be positive. There was no apparent relationship

between the degree of malignancy of the primary lesion and the maximum size of the largest metastatic node, and no apparent relationship between the degree of malignancy of the primary and the tendency to multiple metastases.

The pathological diagnosis of the primary lesion had no apparent effect upon the location of the metastasis. Since there was a high incidence of high grade malignancy of the primary in this series of patients on whom neck dissection was performed, and since the greater the degree of malignancy the more likely that dissected nodes would be positive, we feel that a patient with a high grade primary lesion should be watched more carefully for cervical metastasis than one with a low grade lesion.

Site of Primary Lesion.

Variations in the site of the primary lesion had no apparent effect on whether palpable nodes contained malignant cells. There was also no apparent effect upon the maximum size of the metastatic nodes. Regarding number of nodes, however, cordal-subglottic lesions had the least number of nodes per case; vestibular lesions accounted for the greatest number of nodes per case, and supravestibular and hypopharyngeal lesions following in that order.

Laryngeal and hypopharyngeal lesions metastasized predominantly to the upper jugular and middle jugular nodes in this series as in others.[8] There appeared to be a higher incidence of involvement of the middle jugular nodes in hypopharyngeal lesions. Extension to the lower jugular nodes was fairly evenly distributed with somewhat less tendency of the cordal-subglottic lesion to metastasize to this area. As might be expected from their locations, supravestibular lesions accounted for the greatest proportion of metastasis to the submaxillary region, and hypopharyngeal lesions were responsible for most of the metastases to supraclavicular nodes. Vestibular lesions accounted for two-thirds of the nodes involving the region of the spinal accessory nerve, and in all 223 neck specimens only one positive node was found in the submental region.

Site and Location of Primary Node.

In studying the metastatic nodes it is of interest to note the location of the secondary node in relation to the position of the first node. If the primary node appeared in the upper jugular region, the subsequent nodes were in the middle and lower jugular areas as expected. If the primary node was in the middle jugular chain, however, the secondary node was more apt to be in the upper jugular rather than the lower jugular region.

In 99 cases in which the metastasis appeared clinically as a solitary mass, it was found by dissection of the specimen to be a group of nodes in one place rather than a single node in 31 cases.

We also found that there was a strong tendency for the number of positive nodes to increase as the size of the primary node increased. As the size of the primary node increased the number of areas involved in the metastasis also appeared to increas as did the tendency to multiple metastases.

Survival.

Our findings, based on 84 potential three year survivors, yielded some rather surprising findings. The overall survival figures indicated that 25 per cent were living, with no evidence of recurrence three or more years after radical neck dissection. This finding fairly well approximates the cure rate reported by others[1,3,5,6]; however, when this group of survivors is studied more closely, one is dismayed to find that 13 of the 21 survivors showed no evidence of malignancy on microscopic examination of the neck specimen. There were only eight, or 17 per cent, three year survivors in patients in whom the neck specimen showed positive nodes. This salvage rate is woefully small when one considers the morbidity connected with this operation.

In respect to prognosis, the site of the primary lesion appears to have significance. The percentage of patients living with no evidence of recurrence was much higher in those with primary lesions involving the cordal-subglottic and vestibular regions than in those with the primary site involving

the supravestibular and hypopharyngeal regions; in fact, the supravestibular lesions accounted for the greatest number of patients dead of disease within three years and thus appear to carry the worst prognosis. Patients in whom the primary lesion involved the cordal-subglottic and vestibular regions had better than a fifty-fifty chance of survival. As noted before, it is very encouraging to find five three year survivors of 22 patients in whom the primary lesion involved the hypopharyngeal area. Most of these patients had large lesions spreading on to the epiglottis and the base of the tongue or over into the endolarynx. This finding would appear to aid and abet the recent tendency of attempting a surgical cure of these hypopharyngeal lesions which were previously relegated to palliative X-ray therapy.

There appeared to be little difference in the three year figures between the patients living without recurrence and those dead of disease, whether the operation was performed one week after detection of metastasis or four weeks following such detection.

In the study of survival of these patients, it was apparent that the more positive nodes there are per case, the lower the survival rate. In an analysis of the factors related to the number of positive nodes per case we found two main variables: these were the site of the primary lesion, and the maximum size of positive nodes. A study of these variables shows that cordal-subglottic lesions tend to have fewer nodes per case, and vestibular lesions had the most nodes per case, ranking ahead of supravestibular and hypopharyngeal lesions in that order; and that the larger the primary node is the more likely there will be multiple metastases. In view of the fact that these variables appeared to influence the number of positive nodes per case, and, therefore, the prognosis, it was necessary to determine whether there was any correlation between these factors. A careful study of the data failed to reveal any correlation between the site of the primary lesion and the maximum size of the primary node; therefore, it would appear that the site of the primary lesion and the maximum size of the primary node influence the number of positive nodes per case independently of each other, and,

therefore, by their effect on multiplicity of metastases have an effect upon prognosis.

Since the survival rate is affected by the number of nodes, and since the umber of nodes per case appears to be related to the site of the primary lesion and the maximum size of the primary node, one would expect that the survival rate would be related to these same factors. Indeed, in this study the survival rate did appear to be related to the site of the primary lesion; however, no such relationship could be demonstrated between the maximum size of the primary node and the survival rate in these 84 patients when the *actual* size of the primary node was used. In a previous study of 75 patients by one of us (G.F.R.), there appeared to be a clear relationship between the maximum size of the primary node and the survival rate when the preoperative *estimated* size of the primary node was used.[7] The size of the sample in the present study and in the previous study is virtually the same regarding survival. Since we had originally used two year survival figures, we compared the node size with the two year potential survivors in this study and again found no difference in survival.

At the present writing, we are unable to account for this difference in our two series, but we feel that difficulties in estimating node size accurately and in differentiating a solitary node from several nodes in one place may explain this difference. We still feel that the estimated size is important in prognosis because of our clinical impressions, the findings of the previous study, and the indirect relationship between actual node size and survival demonstrated in this study.

Although we have compiled many facts of interest, the most important finding to us is the overall three year survival rate of only 17 per cent in those cases with positive nodes. This survival rate is woefully inadequate even for salvage surgery, but is probably an unfair indictment of neck dissection as a curative procedure. Most likely the survival rate in this series is low, because we were relatively unselective in choosing patients for surgery during the period of this study. The operation was offered to practically all patients with cervical metastasis in the *hope* of affecting a cure. Perhaps this

philosophy is wrong. Perhaps we should be more selective in our choice of patients for radical neck dissection.

As a result of the present study we feel that the prognosis in these cases is affected by the site of the primary lesion and the number and apparent size of the metastatic nodes, and that the decision to advise radical neck dissection should be tempered by these factors. Perhaps in this way we can more intelligently select patients in whom the potential benefit adequately justifies the operation.

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ELEVENTH CONGRESS OF THE INTERNATIONAL ASSOCIATION OF LOGOPEDICS AND PHONIATRICS.

Those unable to attend this Congress but who would like to secure a copy of the Report of the Proceedings can obtain it at the reduced rate of *seventeen shilling and sixpence* (17/6) by ordering it in advance, enclosing payment, from the Secretary, before August, 1959. Secretary's address: Peggy Carter, L.C.S.T., 46, Canonbury Square, London, N. 1.

THE DIAGNOSIS AND TREATMENT OF FACIAL PARALYSIS, SECONDARY TO BASAL SKULL FRACTURE.*

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Facial paralysis is a not uncommon finding in basal skull fracture. Grove,[1] in an exhaustive study of 211 patients with skull fracture, found that 29 had facial paralysis, an incidence of 14 per cent of all skull fractures, and an incidence of 18 per cent of his series in which the fracture involved the temporal bone. Fractures of the temporal bone are usually of two types: longitudinal and transverse. Longitudinal fractures of the petrous portion of the temporal bone are roughly three times as frequent as are transverse fractures. Longitudinal fractures usually extend from the squama across the posterior aspect of the osseous external auditory canal, across the tegmen, and along the anterior surface of the petrous pyramid. Facial paralysis occurs in longitudinal fractures in 10 to 18 per cent of patients. Bleeding from the ear, conductive deafness, and a visible fracture line in the external auditory canal are common findings in longitudinal fractures. Transverse fractures of the petrous pyramid are more likely to be fatal, and cause facial paralysis in 30 per cent to 50 per cent of patients. Nerve deafness is not uncommon in transverse fractures; hemotympanum may occur, but tears of the tympanic membrane, bleeding from the ear, and a visible fracture of the ear canal are not seen.

A third type of fracture involving only the mastoid process has been reported, but is rare.[2]

Facial paralysis incident to fracture of the temporal bone may be immediate or may be delayed and not develop until several days after the injury.

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Spontaneous recovery of function appears to be the usual outcome in facial paralysis associated with skull fracture. Eighty per cent of Grove's cases recovered spontaneously, and Kettel[3] states that 75 per cent of the immediate type recover completely, 15 per cent show partial recovery, and 10 per cent remain completely paralyzed.

Decompression of the facial nerve in the temporal bone in paralysis secondary to temporal bone fracture, has been reported by: Cawthorne,[4] Farrior and Caldwell,[5] Behrman,[6] Kettel,[3] Maxwell and Magielski,[7] Feinmesser,[8] and others. The patients operated upon by these authors presented fractures of the longitudinal type, and in most incidences the nerve was found to be damaged by the fracture at or near its pyramidal segment.

Although it is generally conceded that the prognosis for recovery of facial paralysis in closed head injuries is good, recovery does not always occur. Stigmata of degeneration, consisting of weakness or incomplete recovery of the facial muscles, spasmodic tic, and mass movement or synkinesis are common in those patients who do recover function after a reaction of degeneration has occurred.

The facial nerve can be decompressed from the stylomastoid foramen to or near the geniculate ganglion with little morbidity, and without damage to the conduction apparatus. For the above reasons, it is my contention that those patients in whom the fracture is felt to be surgically accessible in the temporal bone, should be operated upon as soon after injury as their general condition permits. In this way it is possible to determine the extent of injury, correct the injury to the nerve, determine any defect of the conduction apparatus, and often prevent a permanent conduction deafness. If reaction of degeneration can be prevented by early decompression, the end-result will certainly be better.

SELECTION OF PATIENTS.

Patients in whom a diagnosis of transverse fractures of the temporal bone is made, should not be operated upon. Those with longitudinal fractures involving the nerve distal to the

geniculate ganglion are ideal candidates for decompression. In general, if lacrimation is suppressed or absent, a lesion of the geniculate ganglion is present,[9] but Feinmesser[8] reports a patient with absent lacrimation in whom the damage to the nerve was in the pyramidal portion. Immediate paralysis indicates a more severe lesion of the nerve, while a delayed paralysis is probably due to hemorrhage in the nerve sheath and edema of the nerve. The former should benefit more from decompression.

TECHNIQUE.

The following technique of facial nerve decompression[10] was employed in four patients with facial paralysis following skull fracture.

A simple mastoidectomy is done, utilizing an endaural incision. The antrum is opened widely, exposing the short process of the incus. The junction of the tympanomastoid suture and the digastric ridge is exposed inferiorly. The posterior and superior aspects of the external auditory canal are thinned, and the middle ear entered with a small bur directed antero-medially at a point just inferior and lateral to the fossa incudis. This opening is enlarged, permitting visualization of the tympanic portion of the facial nerve. The nerve is then decompressed from the cochleariform process to the stylomastoid foramen by removal of bone overlying the postero-lateral aspect of the nerve, with motor driven burs and dental excavators. A neurolysis is then carried out on the entire exposed portion of the nerve, along its posterior aspect so that the anteriolar trunk of the nerve is not damaged. The incision is closed by primary suture without drainage, and without turning a flap.

CASE REPORTS.

Case 1. K.A., a 33-year-old man, sustained a severe head injury in an oil field accident in August, 1956. He was unconscious for several days. Bleeding from the left ear, and complete left facial paralysis were noticed soon after admission to the hospital. Examination on October 14, 1956, revealed complete left facial paralysis of the peripheral type, a fracture line of the posterior wall of the left external auditory canal extending to the annulus, and a conduction deafness in the left ear. Roentgenograms showed a longitudinal fracture of the left temporal bone. He was dis-

oriented and uncooperative. On October 20, 1956, two months after the injury, a left facial nerve decompression was done.

Two fracture lines extended from the squamous through the osseous external auditory canal, and a fragment of the canal was displaced medially into the fallopian canal. The incus was displaced anteromedially. Bone fragments were removed, and the facial nerve decompressed from the cochleariform process to the stylomastoid foramen. The nerve was found to be severely contused in its pyramidal portion. Neurolysis was done, and the incus was rotated into near normal position. Hearing was normal to whisper ten days postoperatively. At last examination, approximately six months after surgery, 75 per cent normal function of the left facial musculature was present. Further follow-up of this patient has not been obtained.

Case 2. M.DeB., a 13-year-old girl, was thrown from a horse on Dec. 31, 1956, receiving a head injury. She was admitted to the hospital, where skull X-rays revealed a linear fracture of the left temporal bone. Bleeding from the left ear was noted. Left facial paralysis began 24 hours after injury and was complete the next day. Examination on Jan. 4, 1957, revealed a fracture along the posterior aspect of the left external auditory canal extending onto the tympanic membrane and hemotympanum. She gave a history of having been deaf in the right ear since mumps at the age of six years. Moderate conduction deafness of the left ear and severe nerve deafness of the right ear were shown by audiometric examination. There was no taste disturbance, and lacrimation was excessive on the left. Response to faradic stimulation was normal on Jan. 5, 1957. Electrical testing was repeated on Jan. 20, and loss of response to faradic stimulation, and an altered response to galvanic stimulation were noted.

Decompression of the left facial nerve was performed on Jan. 23, 1957, three weeks after injury. A well pneumatized mastoid was found to be filled with old blood, granulomatous tissue filled the antrum and aditus ad antrum, a fracture line extended from the squamosa through the posterior aspect of the osseous external auditory canal just below the fossa incudis. Decompression of the nerve and neurolysis were done, and the fallopian canal was found to be fractured in its pyramidal segment. The nerve was not severely damaged. First evidence of return of function was apparent one month postoperatively. Five months after decompression, function was approximately 75 per cent that of normal, and no further improvement was present one year after surgery. She has slight weakness of the lower face and some mass movement is evident.. Hearing is normal in the operated ear.

Case 3. N.C., a 31-year-old man, received a head injury in an automobile accident on Feb. 18, 1957. He gave a history of having bled from his right ear but not his left. Left facial paralysis was noted at the time of injury. Examination on March 15, 1957, revealed complete left facial paralysis of the peripheral type, a dry central perforation of the right tympanic membrane, and hemotympanum on the left. There was no canal fracture visible at otoscopy. There was a moderate conduction deafness on the left, and normal hearing on the right. He denied any disturbance of taste, and lacrimation was normal bilaterally. Skull and mastoid X-rays showed no evidence of fracture of the temporal bone, but fractures of both zygoma were shown. Electrical testing of the left facial nerve showed complete reaction of degeneration.

Six weeks after injury there was no return of function, and on April 4, 1957, a decompression of the left facial nerve was done. The nerve was uncovered from the geniculate ganglion to the stylomastoid foramen, and no evidence of fracture or of injury to the nerve was found. A neurolysis

was done and the nerve appeared normal. He was seen again on Oct. 19, 1957, at which time about 65 per cent normal function of his left facial musculature was observed. His ear healed without difficulty, and his hearing was normal to audiometry.

Case 4. C.P., a 12-year-old boy, was injured in an automobile-motor bicycle collision on June 23, 1957. On admission to the hospital, bleeding from the left ear was noted. Skull X-rays indicated a linear longitudinal fracture of the left parietal and temporal bones. Facial paralysis was first noted three days after injury. Examination on July 1, 1957, revealed a complete left facial paralysis of the peripheral type, and a fracture line along the posterior wall of the left external auditory canal, which extended onto the tympanic membrane. There was blood in the middle ear. Moderate conduction deafness of the left ear was present. There was no taste disturbance and lacrimation was normal on the left. Electrical studies were not done. On July 10, 1957, 17 days after injury, a left facial nerve decompression was done. A fracture line was found which extended across the squamosa and mastoid process through the external auditory canal into the tympanic membrane, just below the notch of Rivinus. A fragment of the external auditory canal was found displaced medially into the fallopian canal in its pyramidal portion. Decompression was carried out from just below the geniculate ganglion to the stylomastoid foramen, and a neurolysis was done. The nerve appeared contused but not severed.

Improvement in facial function was evident 48 hours postoperatively. Nine days after surgery he was estimated to have a 90 per cent return of function. He was next seen on Sept. 12, 1957, at which time his facial function was entirely normal, with no stigmata of degeneration, and an audiogram revealed a normal air conduction threshold in the operated ear.

SUMMARY AND CONCLUSIONS.

Facial nerve decompression was carried out on four patients with facial paralysis associated with skull fracture. Three, (Cases numbered 1, 2 and 4) were found to have moderate trauma to the nerve in its tympanic course. In the fourth patient no visible fracture line of the temporal bone was evident on otoscopic or Roentgen examination preoperatively, and no fracture or other pathology was found at operation; however, a transverse fracture through the geniculate area could not be ruled out preoperatively. All patients had normal hearing in the operated ear at last examination. The best functional result was obtained in the patient operated upon the earliest date after injury.

Facial nerve decompression by the technique employed, enables inspection of the nerve and the conduction apparatus without interruption of the ossicular chain and without undue trauma to the patient. It is my opinion that facial nerve decompression should be done on patients with facial paralysis secondary to temporal bone fracture, if the lesion is felt to be

surgically accessible, as soon as the patient's general state of health permits, providing the patient is seen early after injury. Perhaps if this is done a reaction of degeneration can be averted, and a better functional result without synkinesis, weakness or spasmodic tic obtained. Irreversible defects of the sound conduction apparatus may be prevented also.

In those patients who are first seen late after injury, and in whom a reaction of degeneration has occurred, there should be no particular harm in waiting two months before decompression is advised.

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THE PROBLEM OF SINUSITIS IN CHILDREN.*

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Is sinusitis in children much of a problem today? It is apparent to all of us that we no longer see as many seriously ill children as we did before the era of the antimicrobials. Surgery of the sinuses in children is rare today; consequently the problem of sinusitis is primarily one of office treatment and of home management. One is impressed by the relatively few cases of serious complications seen in private practice and even in the outpatient clinics of our hospitals. Children are getting better care from educated and alert parents who rely more and more on the advice of the general practitioner, pediatrician, and otolaryngologist. The widespread use of immunization to an ever larger number of children's diseases, as well as the increased interest in adequate dietary standards and general health measures are factors of great importance in bringing about favorable changes in the character of rhinologic practice; but although the morbidity of sinusitis has unquestionably been reduced in pediatric and adult patients, there has not been a proportionate reduction in their numbers. Many of the problems we see can be traced directly to the misuse or abuse of the very agents that we rely upon for treatment of sinusitis. One of the major problems is the increasing number of staphylococci and other bacteria, which are resistant to the sulfonamides and penicillin. Sometimes this forces upon us the difficult choice of employing antibiotics which are known to cause damage to the neural elements of the ear, to the kidneys, or the hematopoietic system. It is imperative to exercise the greatest discrimination in the use of antibiotics; we must, if possible, determine which one to

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use, how much to use, and when to use it. Perhaps we are using these "wonder drugs" too soon in many cases, before the natural forces of immunity in the host have been mobilized. We are seeing a new type of respiratory disease, characterized by an insidious tendency to chronicity, with a minimum of serious complications, but often with a stubborn resistance to treatment. We see this in the sinuses, the ears, the pharynx, larynx, and tracheobronchial tree.

ANATOMY AND PHYSIOLOGY.

The ethmoids are the only sinuses present at birth[1]; the antrum develops rapidly, with a relatively large ostium which allows for adequate drainage. After the age of three or four years, the antrum floor descends in the maxilla as the facial bones grow, and at this time the ostium becomes smaller. The sphenoids grow slowly and the frontals are the last to develop. All the sinuses achieve their adult size and shape at puberty.

The mucosa of the sinuses is contiguous with that of the nose and is histologically similar. The ciliated epithelium is covered by a thin blanket of mucous which moves toward the ostia. Proetz[2] has stressed the importance of the integrity of the cilia and mucous blanket to the health of the sinuses and nose. Anything which destroys or suppresses this function will reduce the resistance of the mucosa to infection. In infancy, infections of the ethmoids occur with almost every severe cold, and in early childhood the antrum is frequently involved, but most of these infections clear up spontaneously if the general health is good.

BACTERIOLOGY.

Studies by Goldman[3] have shown that the nose is relatively sterile, while potential pathogens are present normally in the nasopharynx. The nose is kept free of pathogens by the natural antibacterial substance, lysozyme which is present in the protective mucous blanket, and the ciliary action which keeps bacteria and foreign substances moving from the nose and sinuses into the nasopharynx. When resistance is lowered, the pathogens from the nasopharynx can invade the nose and sinuses and the tracheobronchial tree. Cultures of the nose

and nasopharynx were made by Goldman, and the nasal cultures were frequently found to be sterile, while those from the nasopharynx were never sterile. The only pathogen found in the nose was the staphylococcus aureus A, while the streptococcus viridans was the organism most commonly found in the nasopharynx. Cultures taken from the nose during infection showed the presence of staphylococcus aureus and pneumococcus to be most frequent, with streptococcus hemolyticus being found occasionally.

Heinberg[4] discusses the problem of resistant bacterial strains, especially the staphylococcus. Whereas the streptococcus formerly headed the list of organisms found in otorhinologic practice, now the staphylococcus is the most common. Heinberg divides staphylococci into the two major groups: saprophytic and pathogenic. The latter are carried constantly on the skin or in the nasopharynx by about 50 per cent of the population and can give rise to serious infection. The coagulase test is the most reliable single, *in vitro* test for the identification of pathogenic staphylococci, which are coagulase positive. After determining that the staphyloccus is coagulase positive, sensitivity tests should be done.

ETIOLOGY.

General Resistance.

Acute sinusitis in children is in most instances a complication of the common cold. Because of this, any factor which results in frequent colds must be considered as a potential cause of sinusitis; thus we see more sinusitis in children during the winter months and among families of lower economic status. Anything which lowers the child's resistance, such as inadequate diet, clothing, or shelter can predispose to sinusitis. In discussing lowered resistance, the existence of hypogammaglobulinemia should be considered. Although there is much to be determined regarding the relationship of Gamma globulin levels to respiratory and other infections, recent studies seem to indicate the probability that reduction in Gamma globulin is an important factor in susceptibility to infection. A recent article in *Lancet* divides agammaglobu-

linemia into congenital and acquired. The former is described as occurring almost entirely in males, and may be a sex-linked recessive trait. The acquired form may be suspected in children of either sex, in whom recurrent bacterial infections have become prominent without other cause, and in whom the Gamma globulin level is below 100 mgm./100 ml. The deficiency is less complete than in the congenital disease, and there may be some antibody formation to a few antigens, but not to most. Enlargement of the spleen and lymph nodes is common.

Impaired Nasal Physiology.

Any abnormalities which result in inadequate ventilation and stasis of secretions in the nose and sinuses can lead to infection. Of particular importance are hypertrophied adenoids, choanal atresia, deviated nasal septum, and enlarged or deformed turbinates. One of the basic concepts in the treatment of sinusitis is to restore normal nasal physiology, and it is frequently necessary to correct anatomical abnormalities in order to establish adequate ventilation and drainage.

Allergy.

Allergy plays a major role in sinusitis, and must be considered whenever the diagnosis of sinusitis is made. Allergic rhinitis causes edema of the mucosa, an altered mucous blanket and ciliary activity is impaired. Polyps, though uncommon in children, may further interfere with ventilation and drainage. The allergic child will more frequently have a chronic sinusitis which resists treatment unless both the allergy and the infection are treated. Evans[6] quotes Shambaugh, who states that "at least 70 per cent of chronic sinus infections and at least 90 per cent of chronic nasal infections can be shown to have an underlying allergic factor responsible for the chronicity."

Dental Infections.

Infections of the antrum in older children may result from poor dental hygiene, periapical disease, or trauma in extractions. This factor, however, is relatively rare in comparison to maxillary infections in adults.

Barotrauma.

In this modern age, both adults and children are being subjected to sudden and severe changes in barometric pressure. These changes, whether in modern flight or in under water swimming and diving, can cause sinusitis.

SYMPTOMATOLOGY.

Acute Sinusitis.

Younger children are more prone to have systemic reactions such as high fever, toxemia, and severe prostration. In milder cases there may be anorexia, lassitude, or simply a general decrease in the child's well-being and loss of interest in his usual activities. Older children tend to have the same symptoms as adults. In allergic children, we see less of the severe, toxic symptoms and more of the general loss of well-being. Purulent rhinorrhea, nasal obstruction, sore throat and cough are usually present. Many of the younger children cannot or will not blow their nose, and the copious post-nasal drainage finds its way to the tracheobronchial tree, resulting in an annoying cough; loss of appetite and even vomiting may occur if much of the secretion is swallowed. Anosmia may be present, owing to the swelling of the mucosa in the olfactory area. Acute otitis media is a frequent complication.

Chronic Sinusitis.

The most consistent finding in the chronic cases is a history of repeated respiratory infections beginning in the Fall and lasting until Summer. Associated with the typical nasal symptoms there is usually cough and frequent involvement of the ears. In some cases, nasal symptoms are minor, and the chief complaint will be cough. In others, the sinusitis is discovered while attempting to find the cause of recurring secretory otitis.

Because the symptoms vary with the sinus or sinuses involved, this fact may be used to aid in the diagnosis. In ethmoiditis, there is headache between and behind the eyes, or in the temporal region; pressure on the globe of the eye

may be painful. In severe cases with orbital involvement there is a typical lateral displacement of the eye.

In acute antrum infections there is pain and tenderness over the involved maxilla. In chronic maxillary infections, pain and tenderness are less prominent; nasal obstruction on the infected side is usual and purulent rhinorrhea varies from scant to copious. Occasionally an older child may complain of toothache and mistakenly consult a dentist.

The frontal sinus is much less commonly involved than the maxillary and ethmoids; it sometimes accompanies an acute anterior ethmoiditis in older children. There may be tenderness to pressure or percussion over the anterior wall of the sinus, but the floor is more commonly tender to palpation. In severe cases with poor drainage through the naso-frontal duct, the pain may be excruciating. When there is a thin anterior wall or a dehiscence present, the pus may escape from the sinus and form a subperiosteal abscess. Involvement of the orbit usually results in edema of the upper lid and a downward displacement of the eye.

The sphenoid sinus is infected rarely without involvement of the other sinuses, especially the posterior ethmoids. Sphenoiditis may be present without symptoms, and will not be discovered unless it is particularly searched for. Pain may be referred to the forehead or eye on the involved side. Occasionally, edema of the upper lids without proptosis may be seen. Secretions from the sinus drain into the nasopharynx, together with secretions from the posterior ethmoids and cause pharyngitis with thickening of the lateral pharyngeal bands and enlarged posterior cervical nodes.

DIAGNOSIS.

The diagnosis of sinusitis in children, as in adults, must be made with a systematic evaluation of the history, symptomatology and clinical findings. A careful history is most important, to determine the general health of the child with particular reference to the frequency of colds, cough, low-grade fever, nasal obstruction and rhinorrhea. A special inquiry should be made regarding possible allergic factors,

such as a family history of allergy, seasonal hay fever, asthma, feeding problems which may have an allergic basis, and allergic dermatitis.

The examination of the nose should establish the presence of pus, deviation of the nasal septum or evidence of allergy such as paling of the mucosa, watery discharge or nasal polyps. The nasal mucosa should be decongested by a spray or tampon of a vasoconstrictor, such as ¼ per cent phenylephrine hydrochloride (Neosynephrin). Placing a small cotton pack against the middle and inferior turbinates will reduce their size and facilitate location of the source of the pus and inspection of the posterior portion of the nose. A large adenoid mass may be seen blocking the choanae. In some cases, nasopharyngoscopy is useful in locating the source of pus, and inspecting the nasopharynx for adenoids. After the nose has been examined thoroughly, the oropharynx should be examined for postnasal discharge, and its color and consistency noted. In long-standing cases, the lateral bands are usually hypertrophied, and the posterior pharyngeal wall has a pebbly appearance. Purulent secretions are seen along one or both lateral gutters.

In older children, as in adults, transillumination and X-ray are of considerable diagnostic significance. In younger children, because of the relative thickness of bone to airspace and the presence of unerupted teeth, they may not be as helpful.

Nasal secretions should be examined carefully. First, their anatomical source should be determined, that is, whether they are from the anterior or posterior group of sinuses. When the secretion is purulent, it should be smeared, cultured, and tested for sensitivity to the antibiotics.

If allergy is suspected from the history, or eosinophils are found in large numbers in nasal smears, skin tests should be done. Pollen and inhalant tests are quite reliable, but food tests are not. The elimination diets of Rinkel[7] are helpful; the common offenders are wheat, milk, eggs, chocolate, corn and peanuts.

PREVENTION AND TREATMENT.

Because the majority of sinus infections are complications of the common cold, much of the prevention deals with the avoidance of colds. Recent advances in virology are encouraging indeed, but until we have specific, effective vaccines, we must endeavor to raise the child's general resistance and reduce his contacts with others who have respiratory infections. This is admittedly difficult, particularly in children of school age. The child should have a good diet, adequate rest and proper supervision of his physical condition. In this regard, attention should be paid to the importance of chilling and exposure. Children are notoriously inept at recognizing when they are cold. They can get chilled while playing outdoors if improperly dressed, or if they get wet; furthermore, young children who are restless sleepers will often toss their blankets in their sleep and become chilled as a result of exposure if their window is wide open. Parents should be instructed to ventilate the room for a short while when the child goes to sleep, and then close the window for the night. Proetz[2] has pointed out the harmful effects of dryness of the respiratory mucosa. Forced air heat, when not humidified at its source is particularly noxious, not only because of the drying effect, but also because of the house dust it circulates.

Van Alyea[3] stresses the importance of supportive measures in the treatment of sinusitis. The child should be made as comfortable as possible. General prostration, which occurs frequently in small children, should be treated with bed rest, and pain should be relieved by aspirin and hot packs to the face. A vaporizer in the child's room helps to improve the humidity and relieve the dryness of the respiratory tract. The care of these children is usually a cooperative effort with the pediatrician or family physician. They are chiefly concerned with maintenance of the highest possible index of general health in the child. In this respect, the treatment of hypogammaglobulinemia in infection prone children is usually handled by them. We have seen several children with chronic or recurring sinusitis who are under treatment for this deficiency. Although results are not always spectacular, the

maintenance of adequate levels of Gammaglobulin appears to be a valuable adjunct in the overall treatment.

An editorial[9] in "Clinical Proceedings of the Children's Hospital, Washington, D. C." states: "Frequently, a dose of Gammaglobulin in 0.1 gm. per kg. of body weight will afford quite satisfactory protection against repeated infection." This injected Gammaglobulin has a half-life of from 14 to 20 days and probably should be repeated every three or four weeks. Intravenous injection is warned against, as severe hypotensive reactions invariably occur.

Dolowitz and his co-workers[10] studied hypogammaglobulinemia in relation to thyroid function. They reasoned that since thyroid secretion produced an increased lymphocytosis and increased protein metabolism, it might increase the circulating Gammaglobulin. Thus they used sodium liothyronine (Cytomel) which acts on the peripheral tissues, producing immediate results. It is absorbed readily from the gastrointestinal tract and is rapidly cleared from the blood stream; thus the oral route of administration was the most satisfactory as well as the most effective. They found that this drug raised the level of Gammaglobulin and appeared to have beneficial effects in the treatment of a variety of infections. Although the number of cases reported is small, the results are significant enough to warrant further study.

Office Treatment.

Every effort is made to obtain early rapport with the child by handling him gently and explaining every step of the examination and treatment. In most cases adequate cooperation can be obtained; this should be done even if we have to limit the first treatment or two to the simplest of procedures in order to gain his confidence. Our primary concern is to establish good ventilation and drainage. The first step is the shrinking of swollen turbinates by a vasoconstrictor spray or nose drops; after a few minutes a cotton pledget of the vasoconstrictor is placed in the middle meatus, under the middle turbinate, if possible. The pack is allowed to remain in position for several minutes and after removal, spot and mass suction are used to remove thick secretions from the

floor of the nose and middle meatus. Care must be exercised not to use too great an intensity of mass suction, as this is painful and may cause greater congestion and blockage. Following suction, the nose is irrigated with normal saline, using a three ounce Asepto-syringe with curved tip. The patient is instructed to lean his head forward, hold his breath and open his mouth as this is done; large quantities of thick, mucopus can be removed from the nasal cavities by this procedure.

The use of argyrol packs has been decried by many in recent years, because of the inhibitory action on the cilia; nevertheless, we still find them extremely useful. Their chief virtue is to produce a slight irritation of the mucosa which causes hypersecretion and stimulates the drainage of pus from the sinuses, particularly the ethmoids. After the packs are removed, the nose is irrigated with saline as described above. We are aware of the danger of argyria from the abuse of silver preparations, and because of this we do not prescribe argyrol for home use. Under controlled office conditions, however, it is free of danger. Its only objection is the messiness.

The value of the Proetz displacement treatment in infections of the ethmoids and sphenoids should be re-emphasized. The technique is so well known that we do not need to review it here. Children tolerate this procedure very well.

When infections of the antrum do not clear up quickly, irrigation should be done through the natural ostium or by an inferior meatus puncture. Irrigation through the ostium is much less distasteful to the patient, and in our hands about 80 per cent can be irrigated by this method. Our technique of antrum irrigation has been described by one of us[11] in a previous publication, but will be reviewed briefly. A small, cotton-tipped wire applicator is dipped in 5 per cent Cyclaine mixed with 1/1000 epinephrine in the proportion of five to one. This is placed at the site of puncture or the natural osteum for several minutes, and repeated two or three times until firm pressure elicits no pain. By this method, necessary analgesia can be achieved at the desired spot without massive exposure of the nasal membranes to the anesthetic agent. So far, we have seen no reactions to the Cyclaine. When the

natural osteum cannot be entered easily we prefer not to puncture the middle meatus.

For the inferior meatus puncture, we use the Lichtwicz needle. After it has been passed into the antrum, a wire stylet is passed through the needle. This dislodges any spicules of bone which may occasionally obstruct the lumen; by having the length of the stylet marked, it can be used as a guide to determine how far the tip of the needle is from the walls of the sinus.

Whether the antrum is entered through the natural ostium or the inferior meatus, much discomfort can be produced by the pressure of the irrigating solution; therefore, it is important that only moderate pressure be used, not only with the solution, but also with the air that is blown in after the irrigation. Air should never be used if there has not been a free flow of the solution. The possibility of serious air embolism must not be forgotten. The instillation of 2 cc. of an antibiotic vasoconstrictor solution seems to be helpful in some of the more chronic cases.

HOME TREATMENT.

The home treatment, as the office treatment, rests on the principle of establishing drainage. This is aided by the use of plain vasoconstrictors, or by one of the nasal sprays that combine vasoconstrictor, antihistamine and antibiotic or Cortisone. These combination sprays seem to be more effective than the vasoconstrictor alone, particularly in allergic cases; however, with the use of any drug that is instilled in the nose, it is important to caution the parent against overuse. In their natural anxiety to open a blocked nasal passage, the drops or spray may be used too often, and the dangers of this must be emphasized. Oral decongestants are also helpful when nasal obstruction is severe.

Infants and even older children are unable to blow secretions from the nose, and it is important that the parents have some means of removing them. The Davol Infant Nasal Aspirator is a simple, inexpensive device that can be obtained at any pharmacy. It consists of a rubber bulb with a glass nasal

tip which fits snugly in the nostril. Suction is obtained by releasing the bulb while the other nostril is held shut. When the nose is filled with thick secretions, the aspirator can be used before the vasoconstrictor is used, in order to allow the drops or spray to flow into the nasal cavity, and again afterwards to draw out more secretions.

Antibiotic Therapy.

The use of antibiotics has been extremely helpful in the treatment of sinusitis, particularly in acute cases. The choice of antibiotic is best determined by culture and sensitivity tests; however, in the presence of severe infections, antibiotic protection should be afforded immediately; the results of the tests when completed can then be correlated with the therapeutic response to the drug already given. It is not uncommon to find the antibiotic we are using is producing an adequate therapeutic response, yet is listed among those to which the bacteria exhibit strong resistance *in vitro*. Penicillin is no longer our first choice, because of the large number of organisms which are resistant to it; furthermore, allergic reactions are increasingly encountered in previously sensitized patients. In this respect, it seems that topically used penicillin, in nose drops, lozenges and aerosols is particularly prone to sensitize the patients. At the present time we prefer to start our patients on one of the tetracyclines or erythromycin. Chloramphenicol, furadantin, and triacetyloleandomycin are all effective against the staphylococci. When fluid intake can be assured in the older children, the sulfonamides such as sulfasoxasole are often very effective.

Although it is not practical to do routine sensitivity tests on every patient, they should be done in all cases of severe infection, or when there is no response to the antibiotic after 48 to 72 hours. It should be stressed that antibiotic therapy is an adjunct to the well established office procedures, and should not be relied upon as the exclusive treatment.

Allergic Treatment.

Evans[6] emphasizes the importance of differentiating allergic and infectious sinusitis, and states that many cases of allergic

sinusitis can be improved greatly by the simple remedy of stopping all nasal medication and treatment. These allergic noses have been irritated and the symptoms aggravated by the attempt to treat an allergic condition as an infectious sinusitis. When the diagnosis of allergy has been established, the management of the case will depend upon elimination of those foods and other offenders which can be properly controlled and hyposensitization to the house dust and other inhalants which cannot be avoided.

In chronic allergic sinusitis, where polyps are present, it may be necessary to remove them to improve ventilation and drainage. Occasionally, under proper hyposensitization therapy small polyps will disappear. As previously stated, however, polyps are usually less common in allergic children than in adults.

Antihistamines are useful in reducing the rhinorrhea, and in combination with oral decongestants they are particularly effective.

Recognition of allergy, and intelligent allergic management by the rhinologist have been factors of tremendous importance in reducing the morbidity of sinus disease. We are indebted to Hansel for the impetus which he has given to this most important aspect of rhinology.

Radiation Therapy.

Radiation therapy for recurring sinusitis in children was used rather extensively a few years ago; reduction in the size of lymphoid tissue as suggested by Levy[12] would presumably aid in the effort to insure good drainage. In recent years, however, the possibility of radiation hazards has been stressed, principally in connection with the development of thyroid cancer in children. An excellent paper on this subject was presented by Baron[13] to this Society last year. One must agree with his conclusion that unless the etiologic connection of irradiation to thyroid cancer can be disproved, it is unwise to use radiation in children if other forms of therapy are available.

SURGERY.

When conservative management fails to control an infection of the maxillary sinus a window is occasionally necessary. The need for a Caldwell-Luc is extremely rare in children. Orbital complications which do not yield quickly to conservative treatment, require surgical intervention with an external approach, whether they are secondary to ethmoid or frontal sinus infection.

Adenoidectomy is sometimes necessary in order to cure infections of the posterior sinuses. In many of these children we have the added problem of recurring otitis media and the adenoidectomy or adenotonsillectomy is of great value.

COMPLICATIONS.

Before the advent of the sulfonamides and antibiotics, the complications of sinusitis were common and serious. The infection frequently spread to cause an orbital cellulitis, osteomyelitis of the skull, meningitis, cavernous sinus thrombosis or brain abscess. Today, these are very rarely seen. The most common complication is secretory otitis which does not respond to treatment. Another frequent complication is cough which clears up only when the sinusitis has been cured. In others, chronic bronchitis may end in bronchiectasis. Dickey[14] in a discussion of Kartagener's Syndrome, where sinusitis and bronchiectasis are present with *situs inversus*, stresses that treatment for either condition alone is not successful, and, therefore, both must be treated simultaneously. Chronic sinusitis which is resistant to treatment is usually part of the clinical picture in cystic fibrosis of the pancreas, and here again bronchiectasis is a common complication. Most of these children unfortunately do not survive.

SUMMARY.

The successful management of sinusitis in children requires the cooperation of the pediatrician or family physician and the rhinologist. The present conservative approach in the treatment of sinusitis is based upon four important principles:

1. Maintenance of the highest possible level of general health.

2. Establishment of ventilation and drainage.

3. Recogntion and control of allergy.

4. The judicious use of the antimicrobials.

It is encouraging that general acceptance of these principles by pediatricians and rhinologists has resulted in a marked reduction of the serious complications which require radical surgical intervention.

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DESTRUCTIVE LESIONS OF THE TEMPORAL BONE.*

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and
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Destructive temporal bone lesions have been a particular challenge in diagnosis since the advent of our antibiotic-chemotherapeutic era, with the marked diminution in the number of acute coalescent purulent mastoiditis cases. The reduction in the acute infectious mastoids, plus Lempert's endaural approach to the temporal bone, has stimulated a desire to find a better means of diagnosing and treating these lesions which too often can be diagnosed only by means of an exploratory exposure and biopsy of the temporal bone pathology.

History, physical examination, laboratory work and radiological study are the preliminary tools at our disposal in arriving at a diagnosis. The symptoms and signs are not ordinarily pathognomonic of a particular entity. Common symptoms and signs of temporal bone lesions are certainly dependent upon which intrinsic or extrinsic structure is affected. Some common symptoms and signs that arise with destructive temporal bone lesions are:

Otalgia—Mild; severe; intermittent.

Hearing Loss—Conductive; perceptive; mixed.

Otorrhea—Serous mucoid; purulent; bloody.

Tinnitus—Low pitched; high pitched; vascular.

Less common symptoms and signs are:

Infection—Meningitis; cerebritis; brain abscess.

V, VI. Nerve Involvement—Anterior apical lesions.

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VII. Nerve Improvement—Middle ear; mastoid; petrous apex.

VII, VIII. Nerve Involvement—Internal auditory meatus; cerebellopontine angle.

IX, X, XI Nerve Involvement—Jugular foramen.

III, IV, V, VI. Nerve Lesions—Superior orbital fissure; cavernous sinus; sphenoid.

Neurological deficits in combination with any combination of cranial nerves.

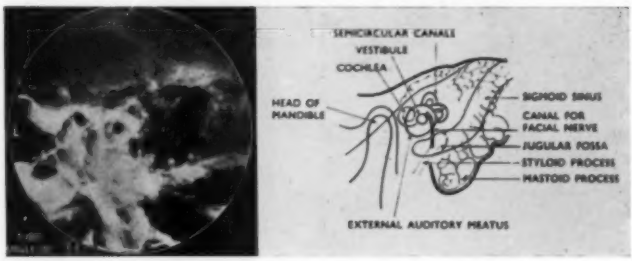

Fig. 1. Lateral mastoid, Law's position.

Radiographic study consists of initial scout films of the Law's position of each mastoid, wherein the internal auditory canal is superimposed on the external auditory canal.

From this view one can ascertain:

1. The size of the mastoid process and its development with cell type integrity;

2. The position of the tegmen with relation to the external auditory canal;

3. The position and integrity of the lateral sinus plate;

4. The integrity of the anterior canal wall and tempero-mandibular joint;

5. The integrity of the hypotympanum;

6. The size of the facial canal, *i.e.*, vertical portion if more K-V is used;

7. A clue as to whether further study of external auditory canal is indicated.

The second scout film used is a modified Towne view, or McMillan view of the petrous apices, which is an anterior posterior projection onto the plate.

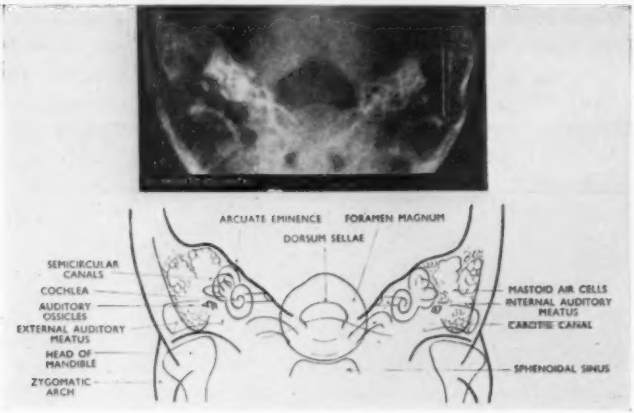

Fig. 2. Fronto-occipetal view, petrous apices.

As all technique factors as to K-V, position, and penetration are equal for the two sides, small differences are more readily seen. From this view, one can usually evaluate, *1.* external auditory canal changes; *2.* internal auditory canal changes; *3.* mastoid antrum changes; *4.* epitympanic changes; *5.* middle ear changes; *6.* petrous apex changes; *7.* mastoid tip changes; *8.* eustachian tube changes; *9.* ossicular destruction at times. Right and left Stenver's views are taken for further internal auditory canal studies. Other studies used when necessary are in stereoscopic views as follows: *1.* mento vertex; *2.* petrous apices through the orbits—posterior, anterior; *3.* modified Towne.

The mento-vertex view permits evaluation of the base of the skull as well as of the temporal bone anatomy. One can as-

sess, *1.* the middle ear; *2.* ossicle position; *3.* foramen lacerum; *4.* basi-occiput; *5.* internal auditory canal; *6.* foramina spinosum and ovale; *7.* pharynx, and *8.* the posterior bony palatal area.

Stenver's view is used for study of the internal auditory canal, the semicircular canals, vestibule and cochlea.

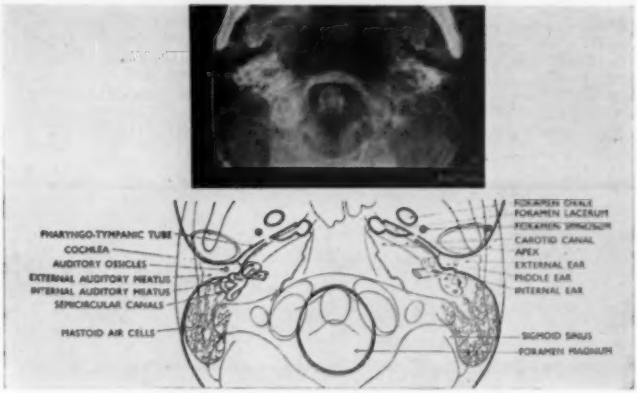

Fig. 3. Base View.

On occasions, a contrast media, such as lipiodol is used to obtain more information about the extent and ramifications of the destroyed area.

The following tables are lesions that we have seen over the years, especially in the past ten years:

TABLE I.

CLASSIFICATION OF DESTRUCTIVE LESIONS OF THE TEMPORAL BONE.

A. CONGENITAL— *1.* Cholesteatoma verum.
B. INFLAMMATORY—*1.* Acute mastoiditis with air cell coalescence; *2.* Pseudo-cholesteatoma; *3.* Osteomyelitis.
C. NEOPLASTIC—*1.* Glomus jugularis; *2.* Neurofibroma; *3.* Acoustic neuroma; *4.* Carcinoma; *5.* Lymphoepithelioma; *6.* Cylindroma; *7.* Carcinoma of the naso-pharynx; *8.* Endothelioma; *9.* Plasmacytoma; *10.* Sarcoma; *11.* Hemangioma; *12.* Irradiation necrosis; *13.* Rhabdo-myosarcoma; *14.* Schwannoma.
D. IDIOPATHIC OR ANATOMIC—*1.* Large sigmoid sinus knee; *2.* Over-expanded air cell; *3.* Xanthomatosis; *4.* Destruction by increased intra-cranial pressure.

TABLE II.

DESTRUCTIVE LESIONS SEEN IN PAST TWENTY YEARS—DUKE HOSPITAL.

Lesion	Number	Lesion	Number
Cholesteatoma verum	1	Endothelioma	1
Acute mastoiditis with coalescence of air cells	16 (since 1946)	Plasmacytoma	1
Pseudo cholesteatoma	213	Sarcoma	1
Glomus jugularis	8	Hemangioma	1
Acoustic neuroma (Neurosurgical service)	54	Irradiation necrosis	1
Neurofibroma	1 (VII nerve)	Rhabdo-myosarcoma	1
Carcinoma (external auditory canal)	21	Schwannoma (VII nerve)	1
Lymphoepithelioma (nasopharynx; involving petrous apex)	6	Large sigmoid sinus knee	2
Cylindroma	3	Over-expanded air cells	1
Carcinoma (nasopharynx; involving petrous apex)	6	Xanthomatosis	3
		Squamous decalcified from increased intracranial pressure	1

CHOLESTEATOMA VERUM.

Duke History C-29465—W.R.B., a white man, age 41, had a long history of pulmonary tuberculosis, which resulted in a left pneumonectomy. His

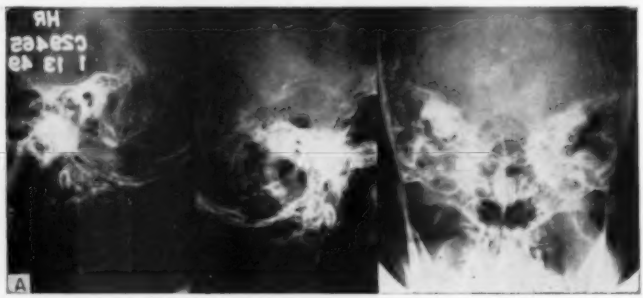

FIG. 1A

ear history dated back three years prior to admission for otorrhea. At examination the drum had a crust over it, extending down to the canal wall. When the crust was removed, a draining fistulous tract could be seen coming through the posterior canal wall lateral to the drum. A catheter was inserted into this area; cholesteatomatous debris was aspirated, and iodized oil put into the cavity. Roentgenograms revealed a destructive lesion, which was inferior to the mastoid and posterior to it, with a very large area of rarefaction in the zygomatic region.

At operation a cholesteatoma verum was found which did not communicate with the mastoid air cells but went posteriorly toward the

cerebellum and posteriorly to the lateral sinus. The mastoid cavity was entered primarily because of the large area of rarefaction in the zygomatic area, and when the mastoid process had been traversed it was found that the area of rarefaction was a clear normal pneumatic cell. The cavity was packed and treated as a radical mastoidectomy postoperatively.

With the history of chronic otorrhea and very little pain, perhaps we should have been able to arrive at this diagnosis, especially after iodized oil was put in, but we did not make the diagnosis until the cavity was opened and its extent verified. The very next patient we saw who had a similar defect and history, did not have the same lesion; hence, it would be difficult to say that from a roentgenological standpoint we could make this diagnosis; it would have to be included in a differential diagnosis.

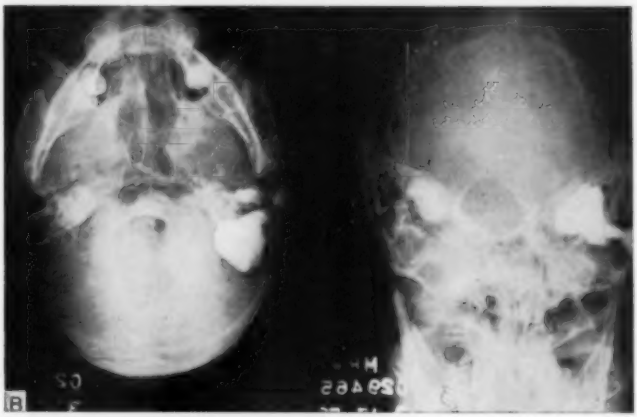

Fig. 1B.

ACUTE MASTOIDITIS WITH COALESCENCE OF CELLS.

Duke History D-9682—R.M.P., age 19 months, a colored female, was admitted to Duke Hospital with a fever and swollen ear of seven days' duration. There had been otorrhea for six days. It had been treated with sweet oil, and when the ear became swollen to a marked degree the mother brought the child to the hospital. The child had received no medical care prior to this time. On admission the child's temperature was 39.5, respiratory rate 36, pulse 150. There was swelling in the retro-auricular area over the right mastoid, and the ear was at a right angle to the head. The canal was filled with a purulent discharge. The swelling extended on to the temporal region. There was no stiffness of the neck nor any other sign of meningeal irritation. Roentgenograms revealed a 1.5 cm. area of decreased density over the mastoid process, showing complete coalescence of cells. On the left there was minimal pneumatization.

A classical retro-auricular incision was made, and a subperiosteal

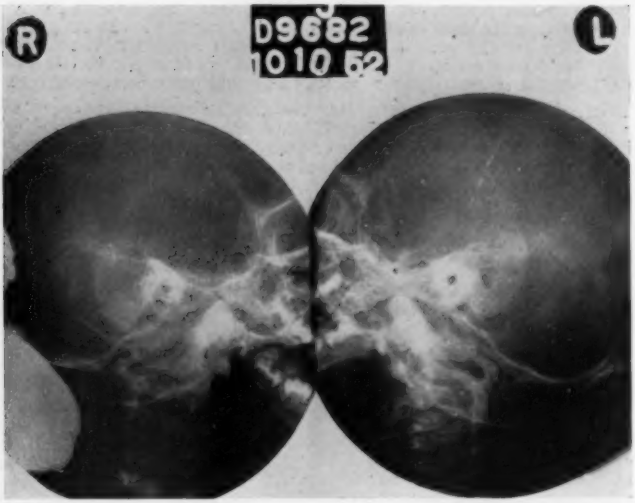

Fig. 2.

abscess was found with erosion of the cortex. Granulation tissue was found in the few mastoid air cells and antrum that were present.

Our interest in reporting this case is that this is the fourth acute mastoid that we have seen at Duke Hospital in six years.

PSUEDO-CHOLESTEATOMA.

Duke History C-82036—J.W.B., age 18, a white male, had had a chronic

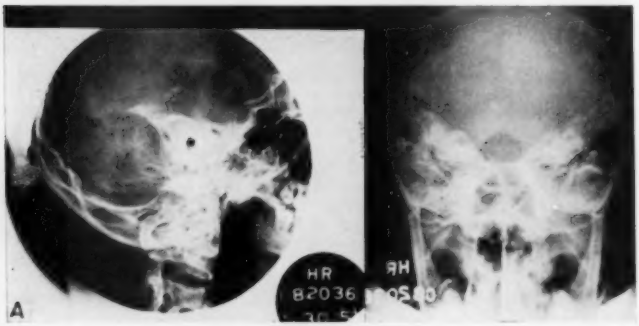

Fig. 3A

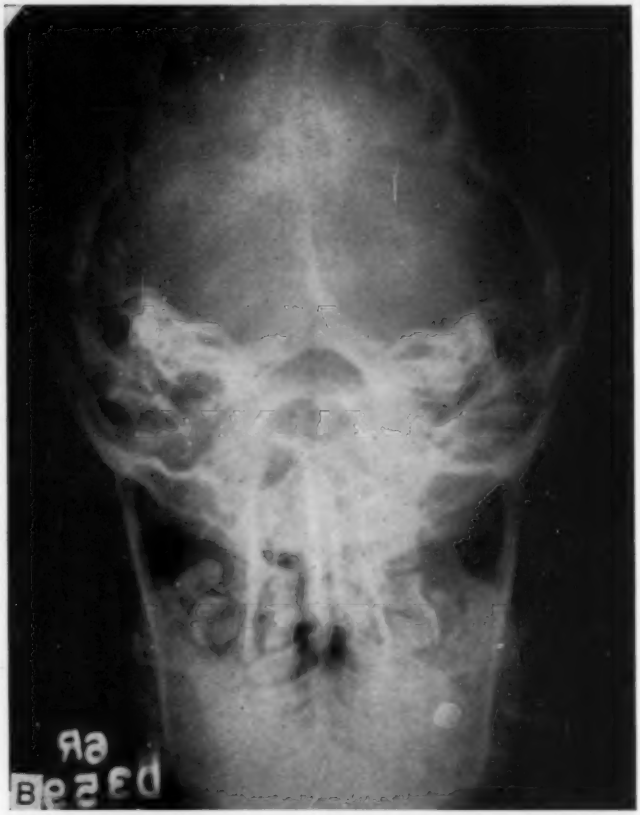

Fig. 3B.

otitis media since the age of 4. He had had polyps removed from his right ear. His left ear quieted down earlier in life, but the right ear never had. Examination revealed the left drum to be scarred but otherwise normal. The right canal was filled with purulent discharge. He had a polyp coming through his central perforation. Roentgenograms of the mastoids revealed a normal looking left mastoid, whereas on the right there was a large destructive lesion, which was interpreted as a pseudo-cholesteatoma.

At operation through an end aural incision, a large cholesteatoma was found which was aspirated, and a routine radical mastoidectomy was

done. The skin of the external auditory canal was laid into the cavity, and the patient was returned to the recovery room.

The pathological diagnosis was pseudo-cholesteatoma.

This is the usual history of otorrhea and minimal otalgia, with the roentgenographic findings.

OSTEOMYELITIS.

Duke History 46896—R.B.C., age 52, a white man, gave a history dating back to 1934, at which time he had an idiopathic osteomyelitis of the skull

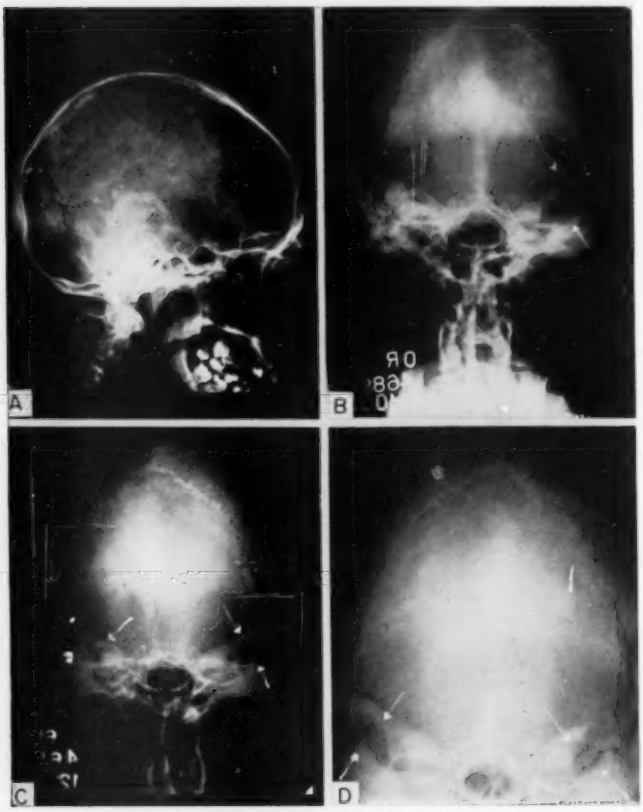

Fig. 4.—A-B-C-D.

with drainage of a temporal abscess. This osteomyelitis of the cranium progressed and then began to regress. Hospitalization was necessitated for almost the entire year of 1935. At this time he had an acute left mastoiditis with operation, that is, a simple mastoidectomy. In 1937 he sequestrated the semi-circular canals and cochlea, which were removed surgically through a retro-auricular incision. During the war years he developed a duodenal ulcer, and in 1948 he was admitted to the hospital with the following findings: For five or six years he had complained bitterly about pain in his right ear, which had become completely deaf, and he had an upper right facial paralysis; he already had a complete left facial paralysis. Hearing was gone on both sides. Examination revealed an otorrhea on the right, and tenderness over the mastoid. Roentgenographic examination serially followed, gives a good picture of this man's course. In brief, he had osteomyelitis that gravitated to both temporal bones, and in sequence sequestrated first his left and then his right cochlea and semi-circular canals, necessitating the removal of both. He is now cured of both his osteomyelitis and his otorrhea; he has no difficulty in walking and communicates very well by pencil and paper. He can talk; but because of his facial paralysis he has difficulty in pronouncing his words, and he finds it is simpler to communicate entirely by writing.

This is a case of osteomyelitis of the skull that settled in both temporal bones, sequestrating both cochleas and semi-circular canals. He has a complete facial paralysis on the left and partial return of function on the right.

GLOMUS JUGULARIS.

Duke History 48008—M.B.P., a white woman, age 32, was first seen at Duke Hospital in 1935 with a history of draining right ear of four months' duration and paralysis on the right side of the face of two weeks' duration. Prior to admission she had a history of tinnitus for two years, but no otalgia except just at the time of admission. She had a long history of right-sided headache, vertigo, nausea, and vomiting. Clinically and by roentgenogram she presented a diagnosis of mastoiditis. The pathological specimen sent to the laboratory was diagnosed as chronic osteomyelitis.

This was the same diagnosis that was given five years later when she was re-operated upon because of a history of recurrent bleeding otorrhea and otalgia.

It was not until nine years later that we made the correct diagnosis of glomus jugularis, which was an unknown entity until Rosenwasser reported the first case in 1945.

Transfusion, surgery, and irradiation resulted in a cure in this case. Irradiation was used to sclerose the vascular element of the tumor.

The history of bleeding, otorrhea, otalgia, and cranial nerve paralysis would now give us a lead that we might be dealing with glomus jugularis. Since we have been looking for these cases we have had four. The roentgenographic picture of destruction in and about the jugular bulb, with secondary involvement of the mastoid, should lead one to suspect or entertain the diagnosis of this benign lesion which can be irradiated if the diagnosis is made. Roentgenograms are of a great deal of value in this entity, plus the seeing a hemorrhagic skin or drum covered mass. Biopsy or removal gives the correct answer.

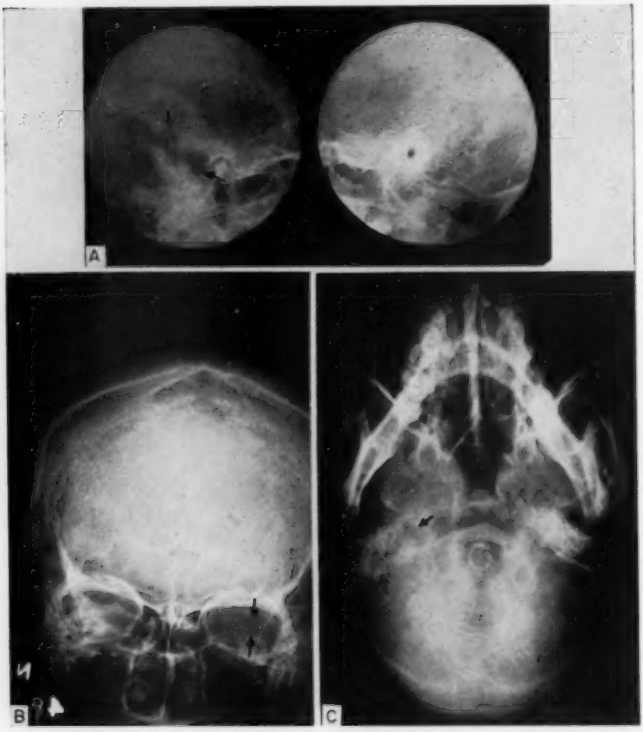

Fig. 5.—A-B-C.

NEUROFIBROMA.

Duke History C-42898—C.S.V., age 13, a white boy, had a history of swelling behind his ear since the age of 4 or 5 months. The swelling progressed very slowly until a history of mumps about five weeks prior to admission to Duke Hospital in 1948. Four weeks before admission he had had an acute swelling following an attack of mumps, and because of destruction around the left temporal bone it was decided that he had a destructive mastoiditis. The mastoid was operated upon. No pus was found, and a diagnosis of lymphangioma was made on biopsy. These slides were unavailable for study.

On examination at our hospital, the patient had a well healed retro-auricular incision. The pinna seemed to detach from the bony skull. The canal was filled with purulent discharge, and it was closed so that the drum could not be seen. The consistency of the mass behind the ear, which extended on into the parotid was doughy and rubbery, not unlike a sac of worms. There was no bruit. Roentgenograms showed a very

smooth absence of bone around the inferior aspects of the mastoid and petrous apex on the left, findings being compatible with an extensive hemangioma.

At operation, a doughy, stringy, beaded fibrous mass was found which pathologically proved to be neurofibroma. His postoperative recovery was uneventful. The follow-up to date has shown no increase in growth of this tumor mass, in spite of the fact that it was not all removed. There was very little pain, very little otorrhea, and no hemorrhage from this ear.

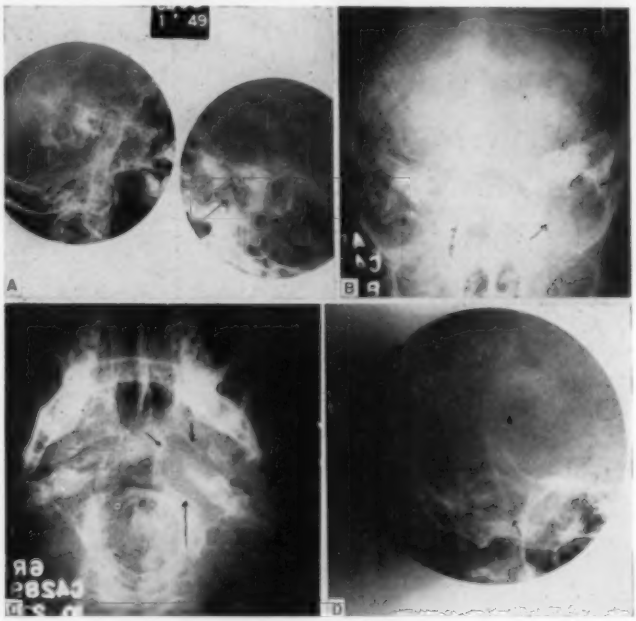

Fig. 6.—A-B-C-D.

Clinically this lesion is progressive and very nearly asymptomatic until a cranial nerve becomes involved. The patients are brought for evaluation, usually because of a deformity about the pinna which seems to have no bony attachment. Roentgenograms reveal smooth pressure erosion with no bone reaction.

Surgically we have been unable to extirpate this lesion because of its ramifications.

ACOUSTIC NEUROMA.

Duke History E-42989—E.G.A., a 58-year-old white male railroad worker

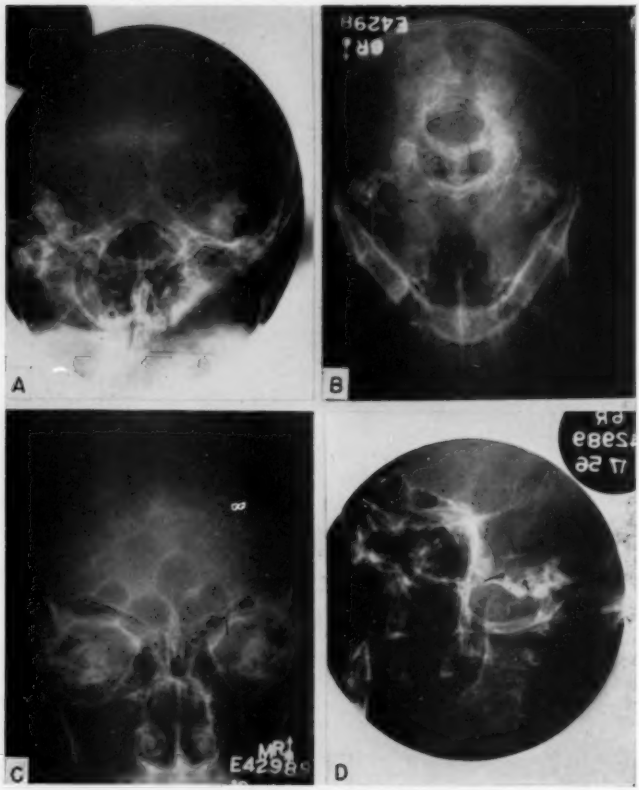

Fig. 7.—A-B-C-D.

came into Duke Hospital on October 16, 1956, with a history of headache, confusion and unsteadiness. His headache was very severe for five or six days prior to admission. There was no history of weakness in any of the extremities or any loss of consciousness. He had some ataxia, the duration of which is not actually known. Physical examination revealed BP 120/78; otherwise negative neurologically, including eye grounds. There was no hearing on the right and an absent response to caloric stimulation on the right. Physical examination was non-contributory except for a spinal fluid pressure of 195 as an opening pressure. His spinal fluid proteins were 150 mgm. per cent.

X-rays revealed erosion and destruction of the right internal acoustic meatus in the mento-vertex, petrous apices and Stenver's views. The neurosurgeons, through a right cerebellar approach, found a right acoustic

neuroma eroding the internal canal. This was removed without undue difficulty. His postoperative course was uneventful, except for the need of a tarsorrhaphy, as he had a VIIth nerve paralysis as the result of cleaning out the internal acoustic meatus.

This case could just as readily have been seen primarily by an otolaryngologist rather than by a neurosurgeon.

CARCINOMA OF THE EXTERNAL AUDITORY CANAL AND MIDDLE EAR.

Duke History A-36089—M.M.B., a white widow, age 64, had a history of otalgia, otorrhea, and swelling in and from her right ear of two years' duration prior to being seen at Duke Hospital. Her system review was essentially negative. The pain was worse at night. The discharge at first was watery and later became thick and yellow. There was apparently no bleeding from the external auditory canal, but she had had bleeding from her pharynx about three months before being seen at this hospital. She also began to have trismus. The right side of her nose became obstructed. The examination was entirely limited to the right ear. The canal was filled with bloody dark exudate, which came from a neoplasm in the external auditory canal, which bled readily. The same type of tumor growth could be seen in the nasopharynx coming from the right eustachian tube and blocking the right posterior choana.

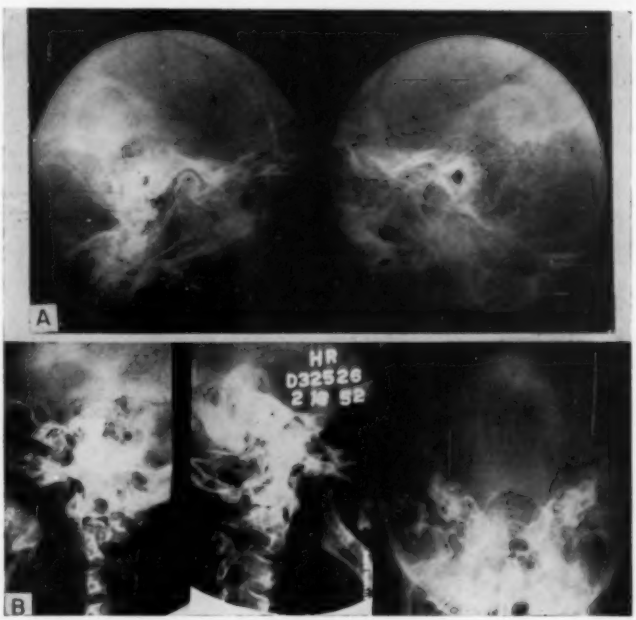

Fig. 8.—A-B.

The tumor mass was biopsied and found to be squamous cell carcinoma. Roentgenograms revealed destruction of all the cells in and around the mastoid. This was probably the result of carcinoma and infection.

The patient was treated with 7800 *r* of X-ray, and she was not seen again after the X-ray therapy had been completed.

This represents a case of carcinoma of the ear with destruction of part of the temporal bone extending down the eustachian tube. The condition was obviously inoperable from the time she was seen, and it was hoped that X-ray therapy might be a mode of treatment. After 7800 *r* to the patient's tissues, the tumor continued to grow, and there is no doubt that she died from the lesion.

LYMPHO-EPITHELIOMA.

Duke History A-150—B.L., a white married woman, age 32, first presented herself at Duke Hospital with a history of otalgia and fluid in the middle ear. The middle ear soon became purulent and revealed roentgenographic changes compatible with mastoiditis without cell destruction. After symptoms of three weeks' duration a simple mastoidectomy was performed. No growth on culture was obtained.

The patient's pain persisted; her jaw became limited in motion, and exophthalmos occurred. Mento vertex plates taken when these signs and symptoms were present revealed destruction of the petrous tip and basi-occiput.

A positive biopsy of lympho-epithelioma was obtained from the nasopharynx, which readily accounted for all the patient's signs and symptoms.

This patient presented a destructive invasive lesion of the nasopharynx, which spread through the foramen lacerum, superior orbital fissure and obstructed the eustachian tube causing her ear symptoms.

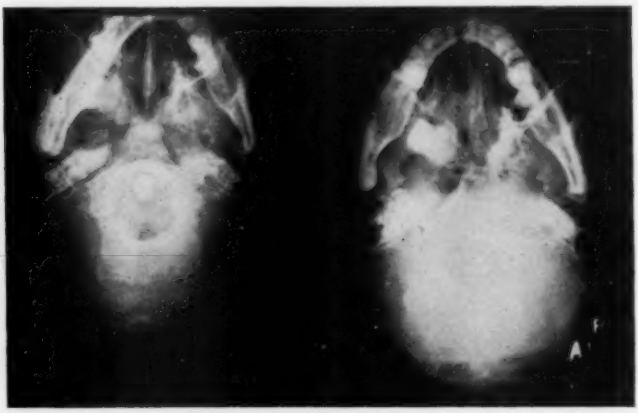

Fig. 9.

CYLINDROMA, NASOPHARYNX.

Duke History C-56448—E.B.B., a white woman, age 48, was first seen at Duke Hospital with a history of pain in her right face which had been present for 18 months. Examination revealed a tender mass with enlargement of the alveolar ridge on the right with bulging into the mouth. The area was fluctuant, and on roentgenogram the lateral wall of the antrum and palate seemed to be gone. We thought that we were dealing with carcinoma, and exploration revealed a hard, rubbery, doughy mass that extended into the antrum. The pathologic diagnosis was cylindroma.

This patient was followed from the Fall of 1949. Because of further extension of this tumor into the nasopharynx and erosion at the base of the skull, the patient was admitted on the neurosurgical service for section of V, IX, X cranial nerves, and I, II, III cervical nerves for pain relief.

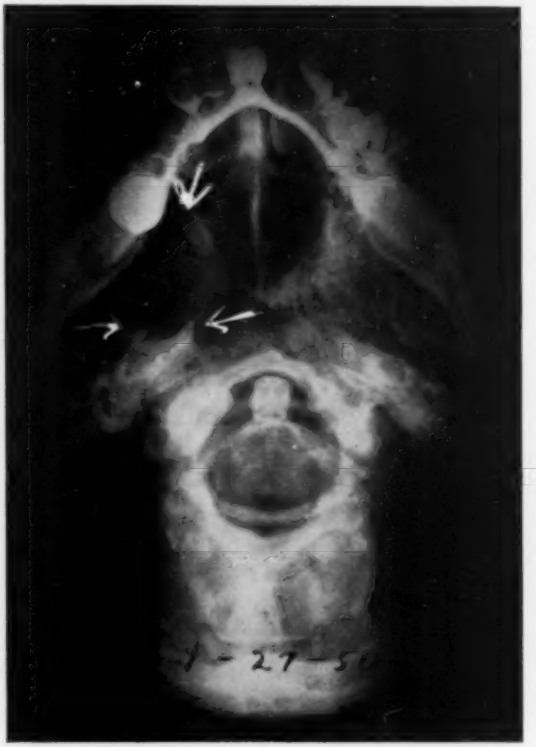

Fig. 10.

The patient died in April, 1950, from intracranial extension of the cylindroma.

Roentgenograms taken in the mento-vertex position showed destruction of the petrous tip along with basi-occiput and portions of the sphenoid bone.

This demonstrates a locally invasive tumor that is progressively invasive if not removed. The petrous apex is destroyed by a contiguous destructive lesion.

ANATOMICAL DEFECT OF THE TEMPORAL BONE.

Duke History B-2594—E.M.P., a 37-year-old white married woman, had a history of a grinding noise in her head of about four years' duration. At times it remained constant and was synchronous with her heart-beat and limited to the right side of her head; at other times it disappeared and there was no sound whatsoever. She had been able to eliminate the sound by digital pressure over her carotid. There had been no headaches, convulsions, sensory or motor disturbances.

The examination was entirely negative with reference to her ears, nose, sinuses, and throat; neurologic examination was also negative. Bruit was heard over the right ear with a stethoscope sometimes; at other times it could not be heard at all. Roentgenographic examination showed a defect over the right sinal dural angle. There was no abnormal vascular pattern in this area, and no change was seen over a period of four years. The question of an arterio-venous aneurysm was entertained, and hence an arteriogram was done, and this was found to be normal.

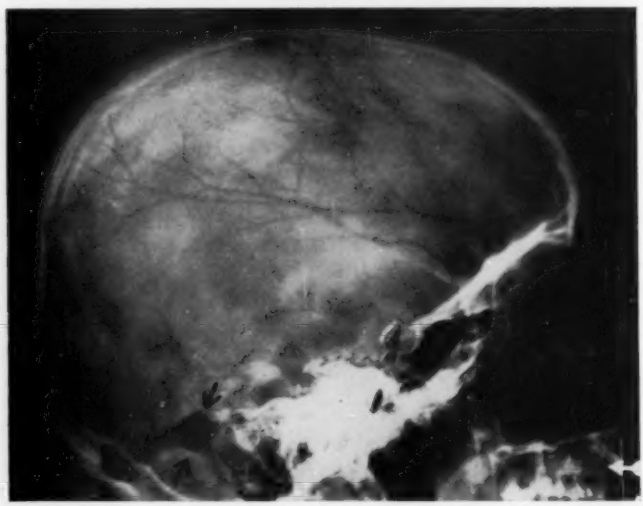

Fig. 11.

The patient was eventually explored. An acute bend in the lateral sinus was found, and when this was released the tinnitus disappeared.

The only symptom was tinnitus, and an apparent anatomical defect in the temporal bone was discovered by roentgenogram. Just why this patient had tinnitus from the lateral sinus, or why it does not occur more often we do not know. We also do not know why it occurred intermittently.

IDIOPATHIC CHANGE.

Duke History B-47314—K.W.S., an 11-year-old white boy, was first seen in 1945 at the age of 4 because of a history of meningitis, for which he

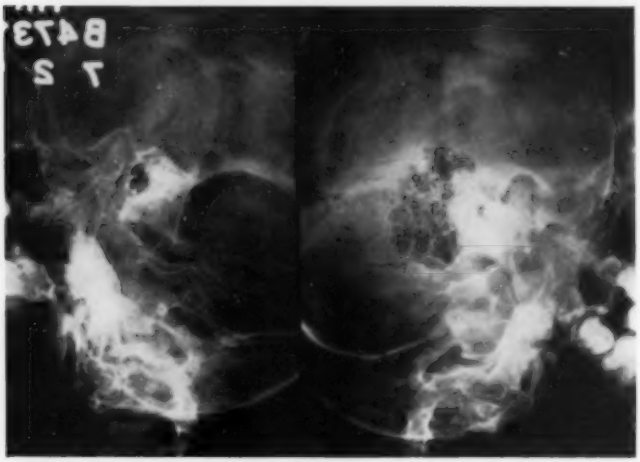

Fig. 12-A.

was hospitalized for a period of 26 days. This meningitis started with an acute infection in his right ear, complicated by mastoiditis, for which he had a simple mastoidectomy. Examination in 1945 revealed no positive physical findings, except that there was questionable bone destruction in the right mastoid. The patient was again seen in July, 1952, because of a facial paralysis that had appeared spontaneously without otorrhea, otalgia, or any other symptoms. Roentgenograms at this time revealed a lesion in the petrous apex, questionable enlargement of the sella, and destruction in and around the mastoid. Because of the complicating factors in this case an encephalogram was done which revealed no abnormality. The right mastoid was explored and a large mastoid cell with normal mucous membrane extending toward the jugular bulb and base of the skull was found. The facial nerve was lying uncovered with new bone formation pressing on it. The nerve was decompressed; but it was decided not to explore this petrous apex as the hearing was good, and the child was asymtomatic except for the paralysis.

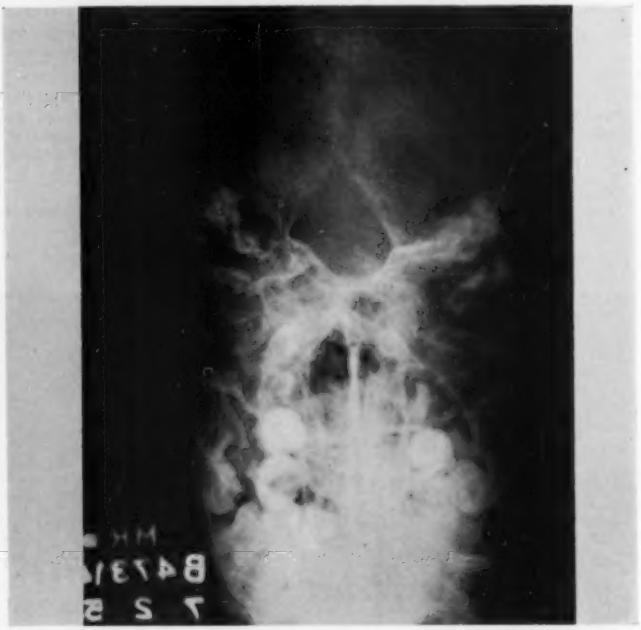

Fig. 12-B.

Just what this boy had, we have not the remotest idea. We feel that his exploration at least advised us that there was no destructive lesion, that is by tumor, and the decompression of the facial nerve has brought back a recovery of his facial function.

MIDLINE CEREBELLAR TUMOR.

Duke History A-7468—M.O., an 18-year-old white single woman presented herself at Duke Hospital with a long history of headaches which had become much worse in the preceding three months. There had been no menstruation for a period of a year. On examination the patient was found to have bilateral papilledema and hyperesthesia over all three branches of the right Vth cranial nerve. There was a questionable right lower facial weakness. The remainder of the neurological examination was negative. Visual fields were normal. Roentgenograms of the skull revealed increased convulsional markings with questionable widening of the sutures. The right squamous temporalis area showed an area of destruction. The dorsum sella was completely destroyed. The patient had ventricular air injection which showed dilatation of the lateral and third ventricle. The aqueduct was displaced upward and dilated. The entire tentorium was pushed up. The impression was that a sub-tentorial tumor had occluded the fourth ventricle.

The patient was explored; a midline cerebellar tumor was found that

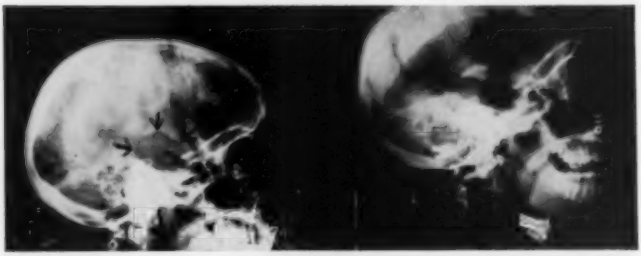

Fig. 13.

had occluded the fourth ventricle, and the pathologic diagnosis was astrocytoma.

This is another type of destruction of a portion of the temporal bone, namely, the squamous portion due to increased intra-cranial pressure. This falls into the realm of the neurosurgeon, but we are including it in our series of cases, as she could just as well have gone to an ear, nose, and throat man with her headaches and received the same diagnosis.

RHABDO-MYOSARCOMA.

Duke History E-68699—F.G.B., a 7-year-old white female child had a

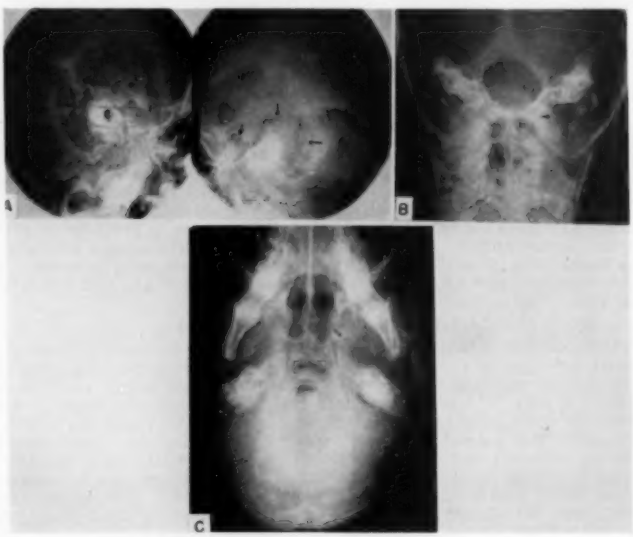

Fig. 14.—A-B-C.

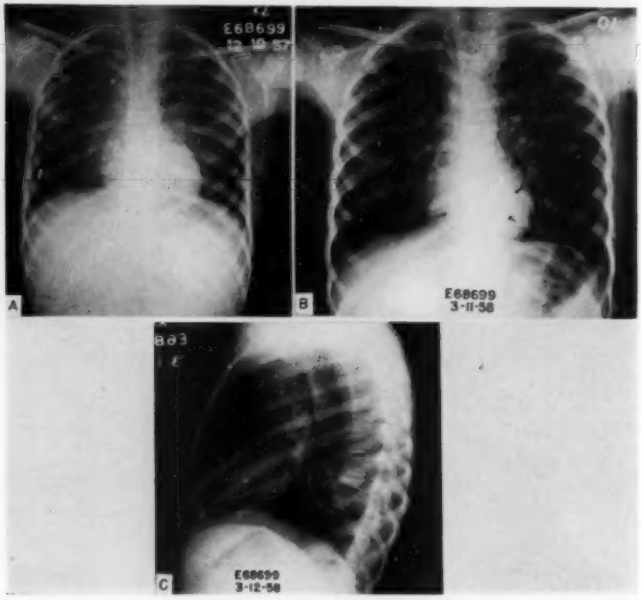

Fig. 15.—A-B-C.

history of a polyp coming from her right ear for three months prior to December 21, 1957. The polyp was removed, only to recur promptly. On three different occasions that the polyp was removed, pathological examination stated it was chronic inflammatory tissue. A radical mastoidectomy was done, only to have a prompt recurrence of this polyp-like material in the external auditory canal and causing a break-down of the retroauricular wound. It was not until the fourth specimen of material was taken that a new diagnosis came back, which was myxoma. The child was admitted to Duke Hospital on December 21, 1957, with this past history of operation and polyp recurrence.

Examination revealed a beginning weakness of the right facial nerve, and two pedunculated hemorrhagic masses could be seen coming from behind her right ear. They were about the size of a ripe fig. Physical examination was entirely negative except for her right ear. X-rays revealed a well done radical mastoid cavity, but there was decalcification along the eustachian tube, the temporal mandibular fossa, and questionably along the foramen lacerum. On the following day, December 22, 1957, an endaural incision was made, and a soft gelatinous mass was removed from the mastoid cavity, and all the bone covering the tegmen, lateral sinus, dural sinal angle, cerebellum, anterior and inferior bony canal walls were removed. The retroauricular incision was closed with interrupted sutures and plain gut. A pressure dressing was put on. The patient withstood the procedure well.

This was followed by a series of X-ray with no local recurrences of this lesion seen. Further study revealed it to be probably a rhabdomyosarcoma. She had a clear chest plate on December 21, 1957, the day of admission, but on March 1, 1958, she was re-admitted to the hospital with metastases to the chest and spinal column. She subsequently died six weeks later from this very malignant sarcoma. Nitrogen mustards were also tried following X-ray, but this was to no avail.

This represents a very destructive, rapidly growing lesion which had all the appearances of benignity under the microscope until rapid regrowth necessitated a more careful study and the tentatively correct diagnosis was arrived at.

COMMENT.

Despite the number of varied destructive lesions that are listed, most of them fall into a category that can be remedied surgically, by irradiation, or both, as the case may be. Fortunately, primary tumors arising in the middle ear are extremely rare, as reported by Furstenberg[1] in 1924. He reported the second carcinoma at this time. His literature review stated: Newhart found only 34 cases of middle ear carcinoma prior to 1917. Gerber stated there is one middle ear cancer to 5000-10,000 of other ear disease. Tod, quoting from the records of London Hospital, where more than 200,000 out-patient visits a year are recorded, encountered one case of middle ear cancer. Fraser[2] in 1930 reported that among 646 cases of diseases of the external meatus seen between 1906 and 1929, there were only three cases of malignant disease (0.464 per cent). In the Royal Infirmary he found that among 6605 cases of the external ear and meatus there were 13 cases of malignant disease, that is 0.197 per cent.

SUMMARY.

1. A series of cases demonstrating destructive lesions of the temporal bone has been demonstrated.

2. Diagnostic means of establishing a diagnosis have been outlined.

3. There are no simple or routine means of establishing a diagnosis or prognosis of a destructive lesion of the temporal bone unless one has a tissue evaluation.

4. The lesions may be (a) congenital, (b). inflammatory, or (c) neoplastic.

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INSTITUTE OF OCCUPATIONAL HEARING LOSS.

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This course presented by Colby College for seven consecutive years is designed to train otologists and others interested in this problem in initiating and conducting hearing conservation programs in noisy industries. In order to allow for maximum individual instruction, course enrollment is limited. The diversity of experience of the faculty members, assembled especially for this course, provides an unusual opportunity for personal consultation on any aspect of occupational hearing loss.

The dates of the course are August 17-22. 1959, and the fee of $200 includes tuition, board and room. Requests for further information should be made to William A. Macomber, Colby College, Waterville, Maine.

MODERN ASPECTS OF RHINOPLASTY.*

C. J. HEINBERG, M.D.,
(By Invitation),

Pensacola, Fla.

For many years little attention was given by the otolaryngologists to any form of rhinoplasty. They viewed this variety of plastic surgery with alarm and allowed the general plastic surgeons to enter a field which should have remained entirely within their specialty. The rhinologist has the fundamental knowledge of the anatomy and physiology which is so necessary to restore altered appearances, correct deformities and to maintain proper function.

Initially rhinoplasty was regarded only as cosmetic surgery. Today, many otolaryngologists have accepted it as a means of also correcting disturbed nasal function. The nose is subject to trauma because of its prominent position, and this has been one of the basic causes of the need for rhinoplasty. Rhinologists have been forced to adopt the new and better methods of treatment of injuries resulting from automobile mishaps, athletic injuries, industrial accidents, and the many other types of nasal injuries.

Because of the universal recognition of its psychologic, physiologic and esthetic significance, rhinoplasty has made much progress. It is important to note that the term rhinoplasty, which is derived from the Greek Rhis, meaning nose, and Plassein, to form, is now used to embrace more than simple cosmetic nasal surgery. Goldman[1] states that a change in the form of the nose or any component part constitutes a rhinoplasty.

The term includes the categories of cosmetic rhinoplasty, which would combine the correction of external nasal deformities for treatment of the frustrated ego, and functional rhinoplasty for the alleviation of impaired nasal function.

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*Read at the Meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., Jan. 23, 1959.

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It is well-known that nasal deformities affect an individual's mental well-being and may result in social insecurity and economic hardship.

It has been suggested that every candidate for cosmetic surgery be asked three questions:

1. Do you sleep well?

2. Do you eat well? and

3. How many days are you absent from work?

If the patient cannot answer these questions satisfactorily, no surgery should be contemplated, and the individual should be referred to a psychiatrist for an evaluation. In a psychiatric study of a series of cases, Linn and Goldman[2] state that with few exceptions, the patients who came for rhinoplasty were clearly ill from a psychiatric point of view. The syndrome they discovered consisted of a constriction of the ego to the point where these persons believed that people looked down on them because of the shape of their noses. Seeing their deformities mirrored each day, these people magnify what they view to the point where it becomes obsessive. According to Schilder[3] each one of us has a mental image of his appearance. When this is displeasing, the mental well-being of the individual is disturbed.

It is quite amazing to observe the immediate reaction of one of these individuals to a well performed rhinoplasty. There is a rapid reorganization of the patient's mental image of himself. The inferiority feelings are replaced with feelings of self confidence. Patients display new warmth, which is so marked that it is evident in their photographs. Much could be said and written of the psychiatric aspects of rhinoplasty. It is most important that the rhinologist recognize occasionally mentally disturbed patients before surgery and employ psychiatric advice. It is these patients as a rule who cause the postoperative headaches for the surgeon; yet, on the other hand, they can be the most satisfied and grateful individuals when handled properly.

It can be readily understood that in our competitive world of today personal appearance plays an important role in the

success of the individual. Persons who have nasal deformities find that these either detract from their economic advancement, or sometimes become their fortune when they capitalize an oddity, as did Jimmy Durante and W. C. Fields.

FUNCTIONAL.

The problem of nasal obstruction has consistently offered a challenge to the rhinologist. Altered functions of breathing and eating cause patients to consult their physicians for relief from these distressing symptoms. Secondary symptoms, such as dry and recurring sore throat; hearing deficiencies from insufficient aeration of the middle ear due to blocked breathing on the convex side of a severely deviated septum; hyperplastic rhinitis and other sequelae also are subjective complaints.

Most of the nasal deformities involving the external nasal pyramid and/or the nasal septum are traumatic in origin. They may be hereditary or developmental.

Kirschner[4] states that injuries to the nose which cause deformities are found in the newborn. They are:

A. Recent, occurring during birth;

B. Prenatal, occurring as a result of forces applied to the nose during intrauterine life.

During delivery the force of the motive power in the physiology of childbirth creates a pressure of almost 40 pounds. This pressure, which is exerted on the nose during the passage of the infant through the birth canal, frequently causes a separation of the cartilaginous portion of the septum from the vomerian groove. This can be easily replaced with the thumb and forefinger and requires no special training or instruments. It is incumbent on all otolaryngologists to request obstetricians to inspect and palpate every infant's nose for septal dislocations, as a part of his routine examination, to determine the presence of nasal abnormalities and any other deformities of the new born.

The prenatal injury is usually more extensive than birth canal injuries, as it usually involves the bony as well as the

cartilaginous pyramid and septum. It results from intrauterine pressure pushing the nose against the mother's bony pelvis during intrauterine life. Cottle and Kirschner[5] agree that instrumentation of these deformities is valueless and that the nose resumes a midline position, or markedly improves, before the infant reaches the age of 3 months.

The nasal bones, lobular cartilages and septum, deserve very careful study in all cases of trauma. This should include all forms of examination, such as inspection, intranasal examination, palpation, and X-ray films. Only by these means can deformities and alteration of nasal physiology be discovered and latent effects be prevented.

When fracture of the nasal pyramid occurs, it is important to correct any deformities for physiological as well as cosmetic reasons. If the upper lateral cartilages are torn, it is necessary that they be repositioned and fixed in place. Cottle[6] states that the ends of the lobular cartilages help as functioning structures of the nose, and that disharmony between the structures of the external nasal pyramid and the internal nose is a cause of nasal illness.

Most nasal fractures occur in the lower thin portion of the nasal bones. It requires a crushing blow to cause a fracture in the upper half, where the bone is thick and firm. Simple fracture can be reduced easily with an elevator or manipulation with a forceps, such as the Walsham. Open reduction is advocated and offers some advantages. This is done by utilizing existing lacerations or by the usual rhinoplastic approach, and with the elevation of the dorsum, fragments can be inspected directly, repositioned under direct vision and hematomas evacuated. Better cosmetic, as well as physiologic results can be achieved. This is particularly true if there is involvement of the septum.

In cosmetic rhinoplasty, the patient seeks the physician, requesting the operation. When there are nasal deformities resulting from injury, and the function can be improved by the procedure as well as appearance, the physician should not hesitate to recommend it.[7] Rhinoplasty is the only procedure for the correction of deformities resulting from old and

neglected fractures of the nasal bones. Restoration of the nasal bones to proper position and realigning of the lobular cartilages can be done solely in this manner.

SEPTAL INJURIES.

Alteration of the nasal septum from the midline is a common sequella of nasal trauma. As reported in a previous presentation,[8] it is my desire to classify septal injuries chronologically as:

1. Prenatal;
2. Birth injuries;
3. Pre-adolescent injuries;
4. Post-adolescent injuries.

New concepts of treatment make this classification important.

As aforementioned, the prenatal injuries are caused by forces applied to the nose during intrauterine life, and no attempt is made to correct these for at least three months after birth.

The birth injuries are simple cartilaginous displacements, and are replaced by either manual or instrumental manipulation of the cartilage back to the midline. Formerly, pre-adolescent injuries to the nasal septum were problems. If immediate replacement and packing in the midline did not correct and latent deflection occurred, the deformity was left alone until full development of the nose had taken place. Most authorities agreed that the period of full nasal development is about 18 years of age. Correction by submucous resection could not be done in these cases, as removal of tissue, as done in the classical surgical procedures of Metzenbaum, Killian or Freer, interfered with the growth and development of the nose by removal of the growth centers, which are supposedly located at the junction of the quadrilateral cartilage, the perpendicular plate of the ethmoid and the vomer. Many surgical procedures were evolved, such as cross-hatching of the quadrilateral cartilage, in the effort to restore blocked

nasal respiration to normal breathing. Jennes[9] remarks that as the septum goes so goes the nose. If the septum is twisted in a child the nose grows the same way.

When the septum is dislocated from the vomerian groove and caudal deviations ensue, not only is nasal respiration impaired, but facial development in the young, is hindered also. For many years, physicians have recognized adenoid facies from nasal airway obstruction, but it is only lately that septal facies has been regarded as a definite entity.

In the developmental process, the external face muscles serve to lengthen the facial skeleton, and the tongue muscles spread the facial structures laterally. Goldman[10] recognized that when the caudal end of the septum was deviated in children the apposition of these muscles was interfered with, and other deformities resulted. These post-adolescence deformities were resultant from trauma in pre-adolescence. He proposed a term.[9] "Maxillofacial Triad" to designate the disfigurements of:

1. Crooked nose—caused by caudal septal deflection;

2. Gothic arch of the palate—dental deformities—Malocclusion;

3. Chin deformities—Microgenia—Micronathia.

Salinger[11] relates that much has been written and said about the septum in recent years. Older procedures have been subjected to modifications, and new methods advocated. It is quite apparent, therefore, that surgery of the septum is not as simple as it seemed to be in the days when we had only the submucous resection, and before the present era of the enthusiasm for rhinoplasty. This is true, and very fortunate. In his enthusiasm for rhinoplasty, Goldman[12] devised a septal technique which lends itself to surgery on the septum of the child. This operation is a combination of rhinoplastic and septal surgical procedures. The dorsum of the nose is uncovered, as in classic rhinoplasty, and transfixion of the septum is done. The septum is thus mobilized and submucous elevation of the mucous membrane, mucoperichondrium and mucoperiostium is accomplished from the caudal end of the

septum.[11] The septal (quadrilateral) cartilage is incised vertically, creating two caudal vertical segments, which are freed at the base by incisions along the nasal spine and maxillary crest, thus breaking the spring of the cartilage. Posterior bony deviations are replaced in the midline by fracturing, if they are present.[17] The caudal segmented portion of the septum is then sandwiched together by mattress sutures, and septocolumellar sutures reattach the septum to the columella. Light packing with vaseline gauze, and external rhinoplastic dressings are applied. Thus the septum is replaced in the midline with no removal of tissue and no interference with growth centers.[18] It is a prophylactic procedure in the prevention of the maxillofacial triad, and restores normal nasal physiology. This technique is also used in surgery of the septum in adults with more acceptance by the patient, and less chance of saddle nose, dropped tip deformities, and columella retraction, which are seen as sequelae to other types of procedures; therefore, any deformity, injury or disease of the nose, should become the responsibility of the rhinologist;[13] it is he who is dedicated to the prevention and treatment of nasal diseases. In rhinoplasty, the otolaryngologic surgeon has a very valuable addition to his armamentarium.

COMMENT.

Just as the speedy jet planes have superseded the early aeroplane of the Wright Brothers, and atomic weapons have replaced the musket, so have new innovations been added to the armamentarium of otolaryngology.

New concepts of the management of nasal injuries and deformities have been presented with special reference to psychiatric aspects, and the correction of nasal pathology in children, to restore normal physiology and prevent post-adolescent sequellae.

It is emphasized that the rhinologist is the more competent specialist to diagnose and manage nasal injuries.

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109 N. Baylen St.

THE EIGHTH BRAZILIAN CONGRESS OF OTOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

The Eighth Brazilian Congress of Otolaryngology and Bronchoesophagology will be held in Porto Alegre, Brazil, the first week of September, 1959. President: Dr. Ivo A. Kuhl, Andradas 1727, Porto Alegre, Brazil.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

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Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960.

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Annual Meeting: October 10, 1959, Belmont Hotel, Chicago, Ill.

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Meeting:

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Meeting:

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Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting: Palmer House, Chicago, Ill., October 11, 1959.

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Secretary-Treasurer: Dr. Homer E. Smith, 508 East South Temple, Salt Lake City, Utah.
Meeting:

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President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 2401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

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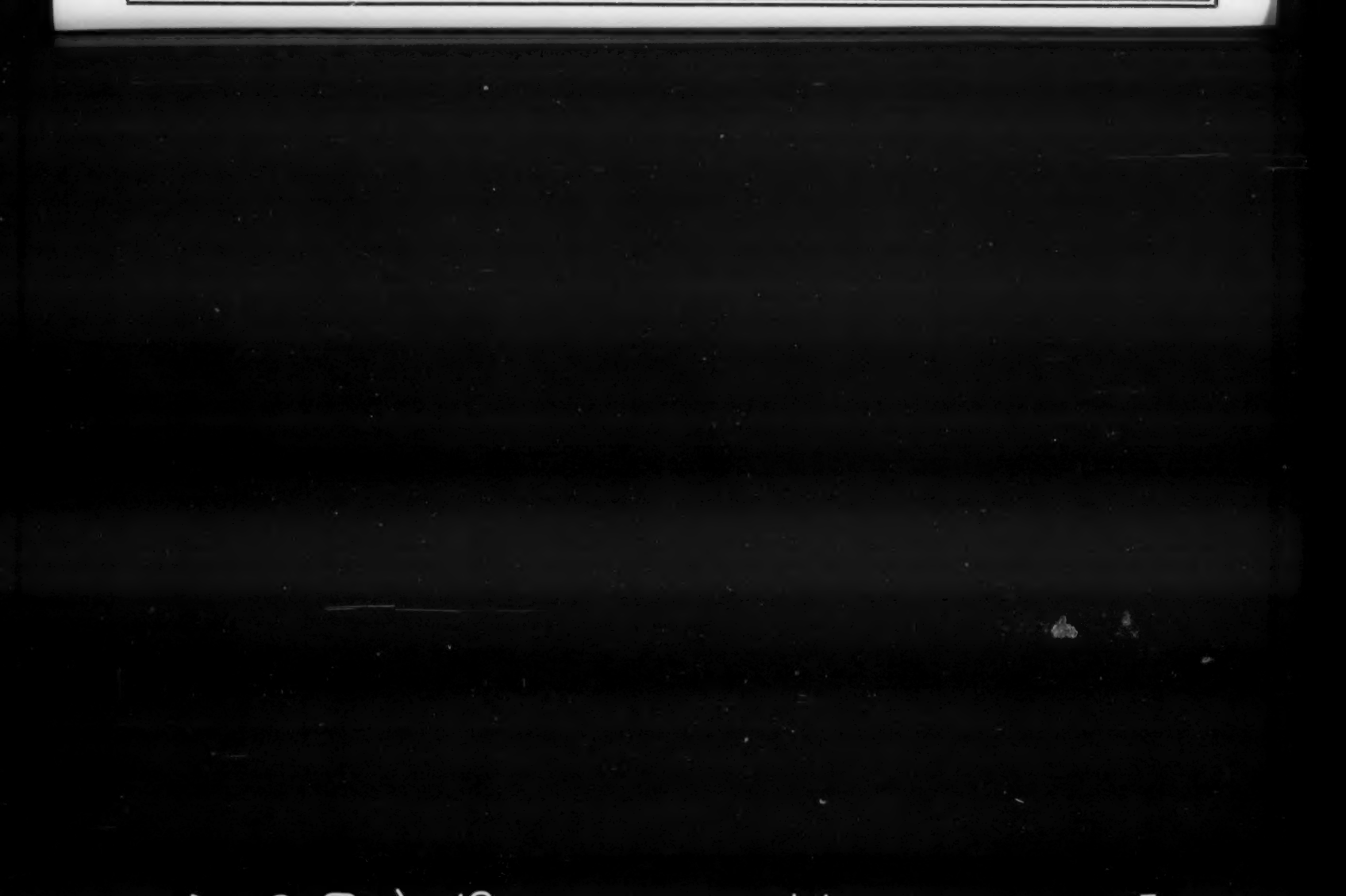

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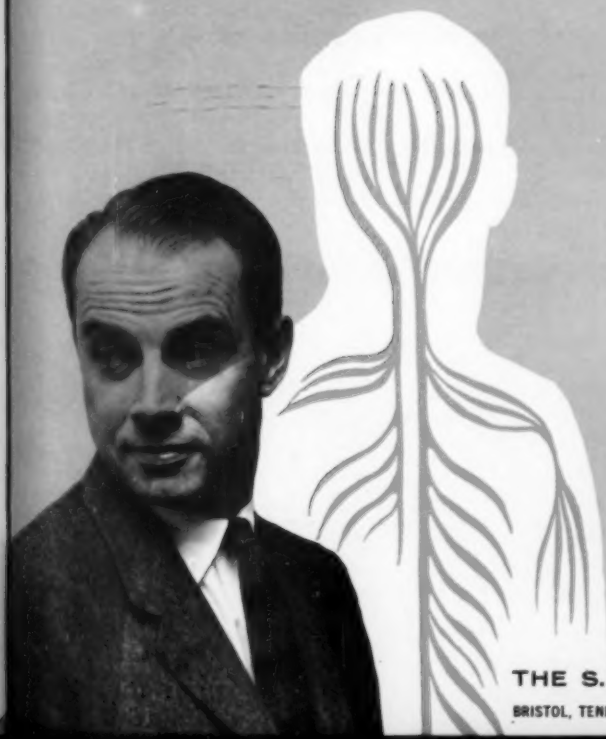

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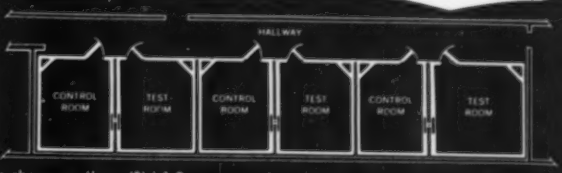

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SURGERY OF THE EAR. By George E. Shambaugh, Jr., Professor of Otolaryngology, Northwestern University Medical School, Evanston, Ill. 669 pages with Index and 326 illustrations (some in color). W. B. Saunders Co., Philadelphia, 1959. Price $27.50.

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